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V. THE FIRST SUCCESSFUL OPERATION FOR GALL-STONES IN ENGLAND

A SUCCESSFUL operation for gall-stones was reported on June 15, 1867, by Dr. Bobbs in the *Indiana Journal of State Medical Science* for 1868 (p. 63), and another by Drs. Musser and Keen in the *Transactions of the College of Physicians of Philadelphia* (1884, Vol. vii, Series iii, p. 265). It was not until Mr. Lawson Tait read "A Case of Cholecystostomy" at a meeting of the Royal Medical and Chirurgical Society on Nov. 11, 1879, that an English surgeon could report a successful case. He says (*Med.-Chir. Trans.*, 1880, lxiii, 16):—

"Dr. Marion Sims attributes to Dr. Handfield Jones, and I think correctly, the merit of first suggesting that the liver and gall-bladder should be included within the field of surgical practice and to my good fortune it has fallen to be the first to follow out Dr. Handfield Jones' idea and Dr. Sims' plan successfully.

"Elizabeth M.—— aet 40 was admitted to the (Birmingham) Hospital (for Women) on August 18th. 1879. on account of an abdominal tumour.

"She had been married eighteen years, had borne six children, her menstruation had always been normal and she had enjoyed perfectly good health until the summer of 1878. At this time she began to suffer severe spasmodic pains in the right side, these being always aggravated by walking or by lifting even slight weights. In September she noticed swelling at the seat of pain and this slowly increased. During last winter her pain became much more intense, her appetite failed, she lost strength and flesh rapidly and on admission she presented an emaciated and almost cachectic appearance. She also suffered at that time from incessant headache and sickness and obstinate constipation. The seat of pain was over the right kidney where there was a heart-shaped tumour, firm and elastic, in which no fluctuation could be detected, and which was extremely tender to the touch. On examination under ether this tumour was found to be perfectly moveable towards each side, indeed, it could be pushed completely across the middle line to the left side. All round it a note of intestinal resonance could be produced. When pushed over to the left side, its heart-like shape became very apparent, and when it lay on the left side of the vertebral column, with its apex directed downwards and to the left, its base evidently retained a connection with the right side.

"A careful examination of the urine gave only negative results, though she spoke vaguely of its having been occasionally dark in colour, muddy and deficient in quantity.

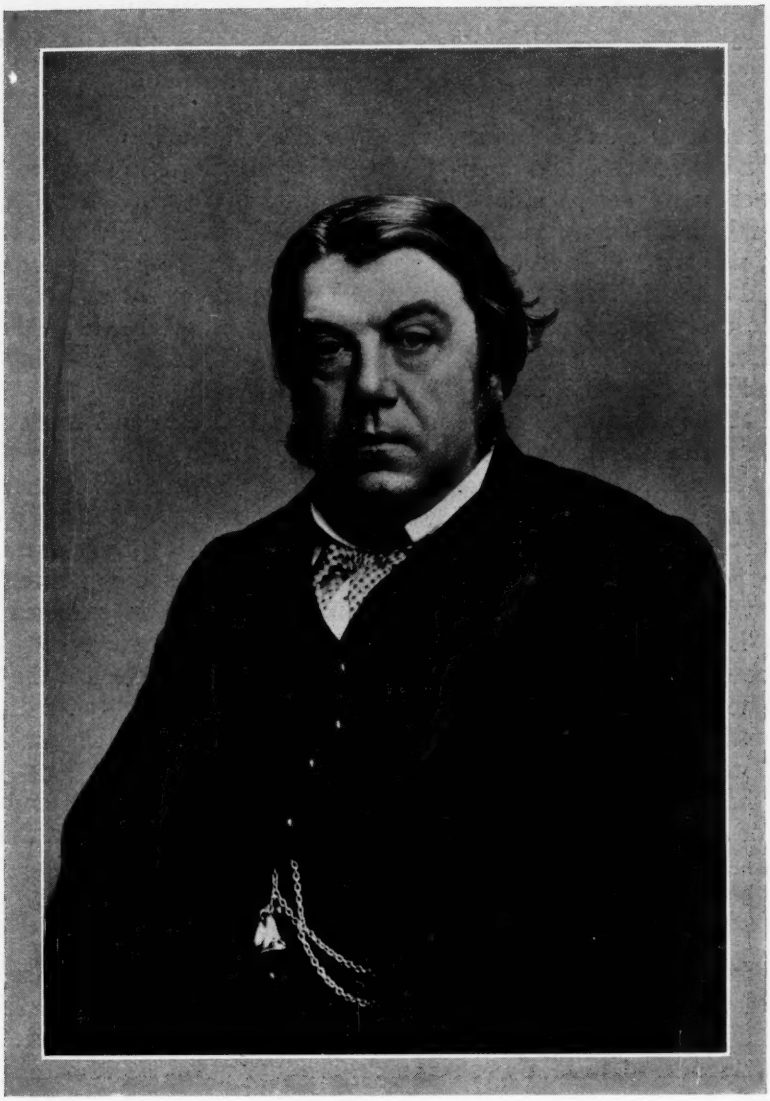
"At the consultation held with my colleague Dr. Edginton upon the case, a variety of suggestions were made for diagnosis, the chief of which were cystic enlargement of a floating kidney, a tumour of the head of the pancreas, and dropsy of the gall-bladder. But no decided diagnosis was attempted and my proposal to open the abdomen and thus ascertain the nature of the tumour was agreed upon.

"On August 23rd. (1879) I opened the abdomen in the middle line, to the extent of four inches, the umbilicus forming the centre of the incision. It then became at once evident that the tumour was a distended gall-bladder. I passed the needle of an aspirator into the apex and drew off a quantity of white starchy-looking fluid, probably amounting to between twelve and fifteen ounces but I cannot speak positively as to its amount, as it was unfortunately thrown away by a nurse immediately after the operation. I then opened the gall-bladder at the point of puncture, so as to admit my finger and came at once upon a large round gall-stone lying loose in the cavity. This I easily removed and on further search I found another of rather larger size, and probably of pear-shape, at the entrance of the duct, impacted in it and evidently the cause of the dropsical distension of the gall-bladder. The removal of this stone was a matter of very great difficulty, in fact it took a very much longer time to effect than all the other steps of the operation put together. From the long narrow funnel-like cavity in which it was lodged and from the mobility of the bladder it was very difficult to seize and when at last I did get hold of it I found it adherent to the mucous surface. I had then to consider the extreme likelihood that in removing this impacted stone I might tear the walls to which it was attached and thus certainly kill my patient. I therefore performed a very careful and protracted lithotrity, chipping little fragments off the stone regularly all over its exposed surface till I had the satisfaction of lifting out its nucleus. I then passed a blade of a pair of fine forceps on each side of it and by a gentle squeeze broke up the remainder and was then enabled to lift it all out. The weight of the stone removed entire is 4·2 grammes and that of the fragments I could gather of the broken stone is 2·9 grammes, but of the latter stone as much again must have been lost on the sponges which were packed into the wound during the process of crushing and upon which I had constantly to wipe my instruments. I washed out the cavity repeatedly and took every precaution that I could to secure that no fragments were left. I then stitched the wound in the gall-bladder to the upper end of the wound in the abdominal walls by continuous sutures, leaving the aperture into the bladder quite open and then I closed the rest of the abdominal opening in the usual way. The operation was performed with complete antiseptic precautions and the anæsthetic employed was ether.

"She rallied from the operation completely in a few hours. I dressed the wound antiseptically the same evening at 11 p.m. and found the dressings stained with healthy bile. In the further progress of the case there is very little to report save that the flow of bile from the wound continued till the 3rd. of September when the dressings were discontinued and zinc ointment was used in their place. The stitches were removed and the wound was completely healed on September 9th. when she began to take solid food, up to that time her diet being restricted to milk and beef tea. On the 14th. she sat up for the first time and on the 30th. she went

home quite restored to health, free from pain and all her former symptoms and having gained at least a stone in weight.

"Looking back upon this case I do not think that a more accurate diagnosis was possible. Fortunately our advanced practice in abdominal surgery makes our



MR. LAWSON TAIT

limited powers of diagnosis in such a case of less importance and I thoroughly agree with Dr. Sims that we should not wait until the approach of almost fatal symptoms puts the diagnosis in unmistakable fashion but that 'we shall make an early exploratory incision ascertain the true nature of the disease and then carry out the surgical treatment that the necessities of the case may demand.'"

PROGRESSIVE POST-OPERATIVE GANGRENE OF SKIN

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In the London Hospital this year, under Dr. Robert Hutchison, there occurred, following a thoracotomy and drainage of an empyema, a case of a remarkable and rare post-operative complication of a slowly progressive gangrenous infection of the skin and subcutaneous tissues. This gradually spread in spite of all therapeutic measures, till, after eight months, it stretched from the occiput to the iliac crest behind and extended anteriorly to involve the whole abdominal surface, and finally caused the death of the patient. Several physicians, surgeons, and others at the London Hospital saw the case, but none of them could remember having met with this remarkable condition before. It was, in fact, only subsequently, quite by chance, that the present writer came upon a similar case in an American journal. This prompted a search into the literature, with a result that it was found that over thirty cases have been reported in America during the last ten years and that the condition has been discussed at at least three Surgical Society meetings; cases also have occurred in Germany, Italy, and Australia. It was realized, firstly, that the condition is not generally known in this country; one case recorded by Hector Scotson[32] last year, which included also a brief summary of eight cases collected from the literature, is the only report that can be found in English journals.* Secondly, and of greater importance, it was realized that failure to recognize the condition may lead to failure, as in our case, to carry out the only effective treatment—namely, excision of the advancing edges, an operation which in almost all the published cases lead to rapid healing of the lesion.

For these reasons it is felt that this case should be recorded and a review included of all the published cases and literature that throw light on this remarkable condition.

THE CONDITION AS AN 'ENTITY'

A study of the cases, 37 of which have been collected in all, at once convinces one that they must be grouped together as an entity and that they constitute a rare post-operative infection of the skin with characteristic and peculiar features demanding a clearly defined line of treatment.

Cullen[1] in 1924 reported a case of "progressive enlarging ulcer of the abdominal wall", which measured 12 in. across, following drainage of an appendicular abscess. It failed to respond to all therapeutic measures, but rapid healing was

* Interest in this condition is increased by the appearance, subsequent to preparing this paper, of a report of a case, only the second to be recorded in this country, in the current number of the BRITISH JOURNAL OF SURGERY (1934, xxii, 392) by Nightingale and Bowden in which they describe progressive gangrene of the skin following laparotomy for a perforated duodenal ulcer. The infection spread steadily till it covered an area 3 × 6 in., when the advancing edges were excised and rapid healing resulted.

eventually brought about by excision of the margins and applying pinch skin-grafts. In the same year Christopher[2] described a case of "severe spreading carbuncular infection of the chest wall" following rib resection and drainage of an empyema. Soreness about the wound developed on the third day, red areas round the stitch-holes on the sixth, and in the third week large pieces of necrotic tissue were removed; the process spread, accompanied by intolerable pain in spite of various local applications, light therapy, and blood transfusions, though the sinus healed up. Eventually after three months it extended from the scapular spine to the iliac crest behind, and anteriorly nearly reached the umbilicus. The advance of the lesion at this stage was successfully checked by excision of the edges, necessitating ten separate débridements under general anæsthesia, and rapid healing resulted.

In 1926 Brewer and Meleney[4, 5] described two cases of "progressive gangrenous infection of the skin" following drainage of appendicular abscesses; each was eventually checked by surgical measures. They recognized that these cases were similar in nature to those of Cullen and Christopher, and Meleney carried out some animal inoculation experiments on the etiology which will be referred to later. The cases were discussed before the American Surgical Association in Detroit,[7] where five similar cases were briefly described by other surgeons present.

After this there appeared at intervals several reports of cases, the majority following drainage of appendix abscesses, all of which were recognized by their authors as being similar in nature to the cases described. All were slowly progressive, carbuncular-like or gangrenous in appearance, with serpiginous outline. They were very painful and peculiarly intractable, being resistant to all ordinary therapeutic measures. The sloughs came away, leaving the base covered with red granulation tissue, which in places showed a tendency to heal, with the appearance of islands of epithelial cells, and the ulcerating undermined edges distinct and separate from the original sinus, which may still be draining or be a healed scar. In nearly all the cases excision and cauterizing of the advancing margins, sometimes in several stages, led to healing, this taking from three to twenty-two months from the date of the original operation to be completed.

At a meeting of the American Medical Association, Philadelphia, in 1931 Lynn[24] read a paper on the subject and included a fresh case of his own. Among those present who joined the discussion were Cullen and Shipley, who previously had published cases, and also Meleney, who had had the unique experience of being able to study and experiment with two typical cases. It was agreed that the similarity between the cases justified them being regarded as a clinical entity and that all the evidence was in favour of a common pathological and etiological basis. The descriptions of twelve further cases which have occurred since then only tend to confirm this conclusion.

INCIDENCE

Although there are only 37 cases which we have been able to collect in the literature, it is stated by some of the authors that the condition is more common than these few literary contributions would lead us to believe. Thus both Ballin[27] and Meleney[37] state that oral communication with other surgeons shows that most of them have seen one or more of these cases without recognizing their importance.

But on the other hand, Meleney's cases[41, 48] of " hæmolytic streptococcal gangrene " which will be discussed later must not be included in this group. It is also difficult to be convinced that one of Ballin's cases,[27] that of an intractable ulcer following an inguinal gland abscess, is of the same type. He excludes climatic bubo in this case by the absence of Leishman-Donovan bodies ; but it has recently been conclusively shown that this condition is due to a filter-passing virus.[44] McDonagh,[38] in a letter commenting on Scotson's case,[32] says he has had experience of several cases, some following appendicectomy, some occurring spontaneously, and some also in the genito-crural region ; that they are due to *B. morgani* and *B. gangrenæ cutis* ; and that they respond to novarsobenzene injections. It will be seen that these cases must be different from the series described here. Before including cases it is necessary to make certain that they showed the remarkably intractable and slowly progressive features which characterize all the cases in the series under consideration. It is no doubt true that many unsuccessfully treated cases have not been recorded and that cases have been reported, owing to the absence of a generic term to describe them, under titles which have not been consulted in our search of the literature.

That the condition is in fact very rare is suggested by the evidence that, though of course abscess formation and cellulitis of the abdominal wall is relatively not an infrequent post-operative complication, gangrene of the chest or abdominal wall is remarkably uncommon in the records of the London Hospital. Thus in the period 1903–33 (excluding cellulitis, gangrene following extravasation of urine, and scrotal gangrene from peri-urethral abscesses) only eight cases have occurred in the chest or abdominal wall which have been described in the notes at any stage as " gangrenous " in appearance. Five of these were acute infections of the tissues round the wound leading to abscess formation, or cellulitis with sloughing and " gangrene " of the overlying tissues (four after drainage of appendicular abscesses, one after abdomino-perineal excision of a rectal carcinoma). One was a case of gas gangrene of the abdominal wall following incision of an ischiorectal abscess secondary to an advanced carcinoma of the rectum, and one followed removal of a stone and drainage of the gall-bladder in a patient with persistent glycosuria in which sloughing of the whole thickness of the abdominal wall took place. In these thirty years one case only can be found in the records of the London Hospital which may have the same etiology as the condition discussed ; it is briefly described here. It is not proposed to include it in the series, as the available evidence is not sufficiently conclusive.

J. H. was operated on in 1925 for a radical cure of an inguinal hernia. " A remarkable gangrene, organism unknown, developed in the wound and spread to involve an area two inches in diameter, and sloughing of the skin and subcutaneous tissues occurred." There are no further notes except that the temperature chart showed a low-grade fever and a pulse-rate averaging 110 to 120 for one month after the operation, on which date the patient left the hospital cured.

NOMENCLATURE

Many different names have been used to describe this condition. Most of the cases during the last three years, including that of Scotson, the first to be reported in this country, have been described under the title " Progressive Post-operative Gangrene of the Skin ", a term first used by Brewer and Meleney[4] in 1924. This

expresses the three characteristics of the lesion and the present case is reported under that heading. Now that the condition has been accepted as an entity it is highly important that some definite term should be universally adopted to describe it, in order to facilitate references and to discriminate it from other skin infections.

CASE REPORT

HISTORY.—A male, aged 44, was admitted to the London Hospital under Dr. Robert Hutchison on Jan. 20, 1934, with the history of an illness which had commenced with a cold and cough nine weeks previously. One week later pain developed on the left side of his chest, sharp in character and made worse by breathing deeply and coughing. The same day he coughed up a little blood, but since then his sputum remained clear. He stayed in bed with the cough, chest pain, and pyrexia for eight weeks, three times having attempted

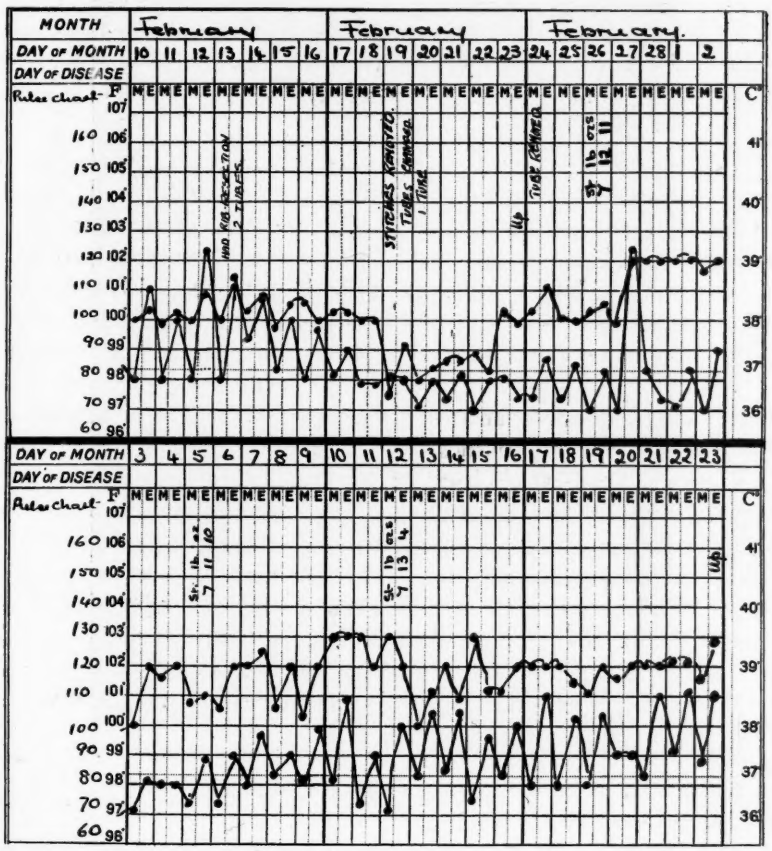

FIG. 441.—Temperature and pulse chart in a case of progressive post-operative gangrene of the skin.

to get up, but being driven back to bed on each occasion by an increase of the symptoms. He was eventually sent up to hospital as his home doctor had found signs of left-sided pleural effusion. Three sputum tests had been reported negative for tubercle bacilli.

Previous Illnesses.—Influenza in 1919. No chest complications. No other illnesses remembered. No history of jaundice. Denied taking alcohol.

Family History.—Two brothers and one sister had acholuric jaundice (investigated by Dr. Janet Vaughan). One brother was well following splenectomy; the other two died

following splenectomy. Father, mother, six other brothers, two other sisters, and four children showed no signs of acholuric jaundice.

ON EXAMINATION.—The patient was found to have an intermittent pyrexia 100° to 102° in the evening and 98° to 99° in the morning. Pulse 100 to 110, respirations 20. He was a thin man, with a good colour; no jaundice; definite clubbing of fingers; heart and arteries normal except for a short systolic apical murmur; blood-pressure 120/80. There was flattening of his left apex, with hollowing above and below the clavicle, and diminished movement of the whole of the left side of his chest. His heart apical impulse was 1½ in. internal to the nipple line and he had a dull percussion note, absent breath-sounds, and tactile vocal fremitus at the left base extending up nearly to the spine of the scapula. The left apex and right lung were clear except for a few transitory crepitations at the right apex.

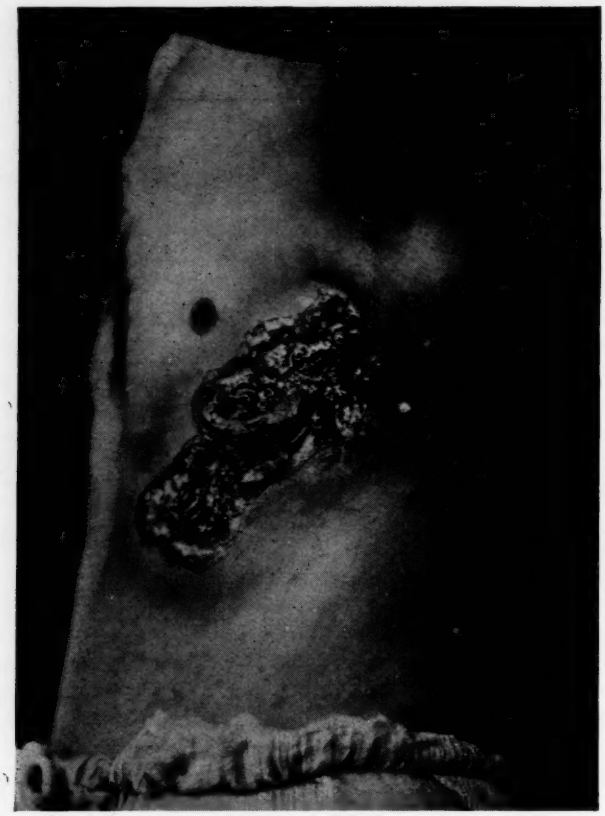

FIG. 442.—The lesion on the fifty-ninth day: antero-lateral view.

Nothing abnormal was found in the abdomen or central nervous system. Urine normal. No tubercle bacilli found in the sputum.

Blood-count (Jan. 22).—Red cells, 3,400,000. Hæmoglobin, 61 per cent. Colour index, 0·9. Leucocytes, 12,400. Polymorphs, 83 per cent. No eosinophils. Slight poikilocytosis and anisocytosis. Moderate polychromatophilia.

Van den Bergh.—Direct, negative. Indirect, less than 0·2 mgrm. per cent. Fragility of red cells: very slightly increased. Complete hæmolysis to 0·35 per cent. Partial hæmolysis to 0·45 per cent. Control's blood: partial hæmolysis to 0·3 to 0·4 per cent.

Jan. 24.—Paracentesis of chest; 16 oz. of thick opaque grey-green pus aspirated, a film of which showed degenerate pus and many Gram-positive cocci in chains. Culture grew non-hæmolytic streptococci. No tubercle bacilli.

Feb. 13.—X-ray of chest. Heart and mediastinum displaced to the right. Left lung showed opacity of the middle and lower zones consistent with the presence of fluid. Right lung, clear.

OPERATION.—A swinging temperature persisted till on Feb. 13 a thoracotomy was performed under local infiltration with 1 per cent novocain. Two inches of the left eighth rib were removed subperiosteally between the posterior axillary and the mid-scapular lines. Two pints of thick pus were evacuated, two tubes inserted, and the tissues stitched closely round to set up a closed drainage system. Empyema wound drained well. Washed out daily with Dakin's solution.

SUBSEQUENT PROGRESS.—On Feb. 19 the edges of the wound were red, inflamed, and bulging round the tubes; stitches and tubes were therefore removed, and one short large-bore tube was inserted and the wound allowed to drain into the dressing. Temperature and pulse-rate were settling well and the general condition was good. By Feb. 20 (7th day after operation) the temperature and pulse-rate were normal and the wound was discharging very little, though the edges were œdematous, red, and tender. By Feb. 23 the patient

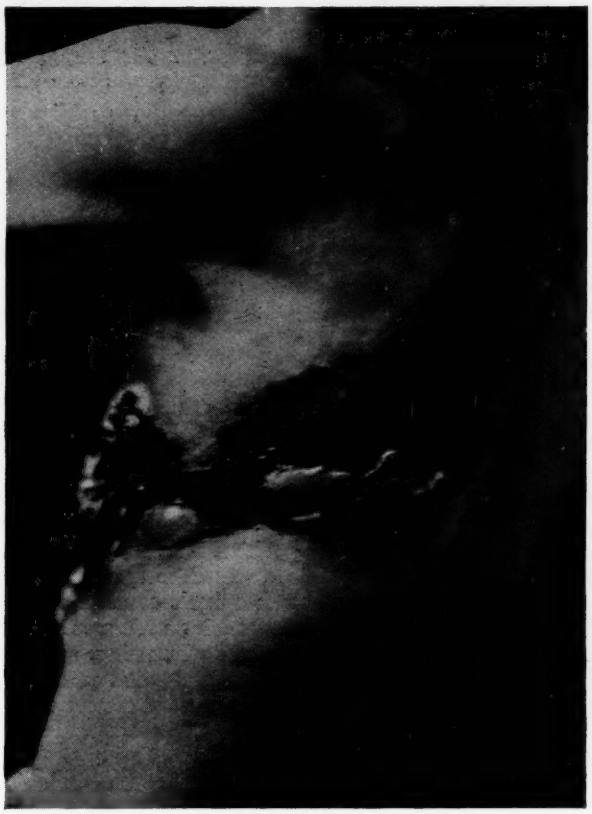

FIG. 443.—The lesion on the fifty-ninth day: postero-lateral view.

appeared so well that he was allowed up for a short period. His pulse-rate, however, began to rise, and on Feb. 27 (14th day) it rose to 120 and was rarely below 110 after this. Meanwhile a swinging pyrexia insidiously started and gradually increased (*Fig.* 441). The wound looked more angry, the œdema and redness began to extend into the surrounding skin, and the edges to slough. Necrosis of the central area took place, and by March 14 (29th day) the wound was described as resembling a large carbuncle. The actual sinus looked healthy and was producing only a little thick discharge. The black, leathery necrotic slough separated in several places, exposing the muscles, which were not affected at any stage and formed the centre of the "carbuncular process"; the edges were tender, raised, serpiginous, and undermined; it was surrounded by a band of red œdematous tissue which was fairly sharply demarcated from the healthy skin. Later the necrotic area separated, leaving the tender

raised margins fairly distinct from the central floor of red granulation tissue covering the muscle layer, which in places showed a tendency to heal.

By March 27 (41st day) the wound stretched from the sternum anteriorly to the edge of the vertebral column posteriorly. The anterior and posterior extremities had acutely inflamed and advancing margins, while the intermediate edges were comparatively healthy and shelving, with outgrowths of epithelium spreading across the base of the ulcerated area. By April 14 (59th day) the process had reached the stage shown in *Figs.* 442, 443, extending beyond the right nipple line anteriorly. The lesion then began to spread upwards and downwards, and slowly progressed till at the end of six months it involved the extensive

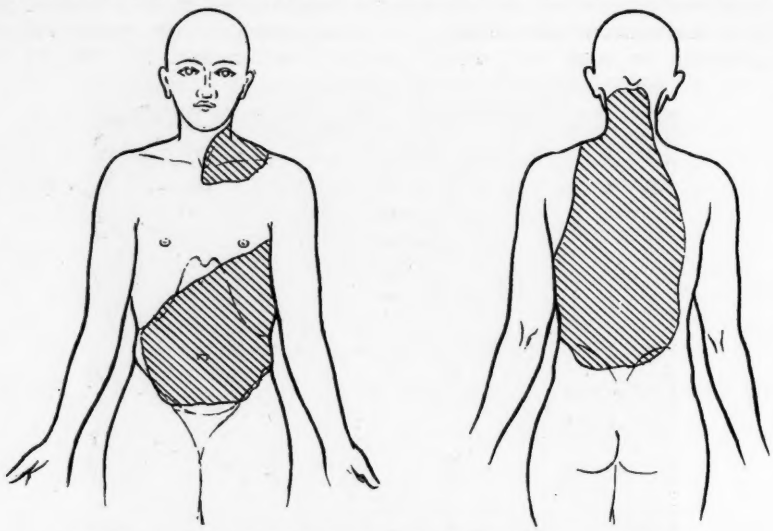

FIG. 444.—Diagram to show extent of lesion at death (thirty-second week).

area shown in *Fig.* 444. The sinus had healed up and there were several islands of epithelium round the site. The edges were exquisitely tender, and great difficulty was experienced with the dressings and finding a comfortable position for the patient to lie in. Apart from pain on pressure and during the dressings there was little spontaneous pain, and sedatives were not required to the same extent as in Christopher's case. In himself the patient was not very ill, but as the process spread and involved such a wide area and the swinging pyrexia continued for so many months he began to show signs of wasting, and during the last two or three weeks he lost his morale and strength, and eventually died quietly on Aug. 27, 194 days (32 weeks) after the operation. His appetite remained good almost to the end.

Blood-counts.—

	March 27	April 3	June 27	July 20
Red cells	4,800,000	4,000,000	4,400,000	4,100,000
Hæmoglobin	77%	66%	60%	65%
Colour index	0·79	0·82	0·68	0·8
White cells	15,000	13,600	9,800	12,600
Polymorphonuclears ..	72%	75·5%	81%	79%
Eosinophils	2%	1·5%	2%	2·5%
Large lymphocytes ..	9%	9·5%	10·5%	10·5%
Small lymphocytes ..	11%	8%	2%	6%
Large hyaline ..	6%	5·5%	4·5%	2%
Basophil	0%	0%	0%	0%

Blood Culture.—Sterile (May 4).

Blood Wassermann Reaction.—Negative (June 12).

Radiograms of Chest.—March 3.—"The condition at the left base is improving. The heart is returning to the left chest. The diaphragm is beginning to move. There is, however, an area of density in the mid-axillary line which might be fluid or might be thickening."

May 20.—"The chest shows no collection of pus now. The whole condition is much clearer". (M. H. Jupe.)

Culture Reports.—April 7.—Pus from sinus: "Streptococci in direct film. Pale and abundant growth of *Staphylococcus aureus*."

April 10.—Non-hæmolytic streptococci. No influenza bacillus. (A. Greaves.)

April 12.—Swab of wound: No true diphtheria bacilli present. Films and cultures show presence of *Staphylococcus aureus* in great abundance.

Sputum.—July 12.—No tubercle bacilli. Chiefly pneumococci with some staphylococci, sarcinæ, and *M. catarrhalis*.

TREATMENT.—

General.—High vitamin diet. Iron, arsenic, strychnine, cod-liver oil, malt, and advita capsules were given by mouth at different stages. Massage to legs.

Local Applications.—Various, including glycerin and magnesium sulphate, orthoform powders.

Sedatives.—Luminal, aspirin, pyramidon, and heroin.

April 13.—36,000 units of diphtheritic antitoxin.

April 14 and 18.—Staphylococcal toxoid (1/60) 0·1 c.c. and 0·3 c.c. respectively: not repeated as pulse-rate and temperature appeared to be higher after the two injections.

April 16.—Light treatment started. Appeared to make condition worse.

June 21–Aug. 10.—Weekly course of autogenous staphylococcal vaccine. No apparent effect.

Blood Transfusions on July 26 and 31 and Aug. 11 and 18. No improvement.

CLINICAL FEATURES

Of the cases collected, 28 were male and 6 were female. The age distribution is shown in the following table.

AGE	CASES	AGE	CASES
18	1	50–60	5
30–40	9	60–65	8
40–50	3	70	1

The operations after which the infection occurred are tabulated below:—

OPERATION	CASES	REFERENCES
Appendix abscess. Drainage ..	20	Cullen,[1] Brewer and Meleney,[4, 5] Porter,[7, 8] Moschovitch,[10] Clinton,[11] Alexander,[12] Mayeda,[13] Gordon,[16] Cole and Heideman,[17] Meleney,[23] Lynn,[24] Warfield,[25] Mitchell,[26] Ballin and Morse,[28] Baker and Terry,[31] Scotson,[32] Meleney,[35] Carol.[36]
Upper abdominal abscess. Drainage. (One probably appendicular,[14] one subhepatic following a previous perforated duodenal ulcer.[33])	3	Brewer and Meleney,[6] Shipley,[14] Meleney.[33]
Simple appendicectomy. No drainage	1	Gillespie.[15]
Purulent cholecystitis. Removal of gall-bladder	1	Porter[9]
Chronic cholelithiasis. Drainage of gall-bladder	1	Freeman[19]
Carcinoma of cæcum. Cæcostomy	1	Meleney[34]
Pelvic inflammation. Oöphorectomy and drainage	1	Borelli[3]
Empyema. Rib resection and drainage	6	Christopher,[2] Poate,[21] Brunsting,[22] Ballin and Morse,[27] Patterson,[30] Stewart-Wallace[36a]
Suppurating epididymitis ..	1	Ballin and Morse[29]
Breast abscess sinus. Drainage ..	1	Probstein and Seelig[18]
Ventral hernia through old cholecystectomy incision. Repair	1	Tennant[20]

It will be seen that of the 37 cases 33 followed drainage of a purulent infection, of which 21 were appendix abscesses and 6 were empyemata.

In about half of the cases the first sign of the new infection is apparent during the first week, and in the majority of the remainder during the second week, the extremes being the first[23] and twenty-second day.[30] In the later group the pyrexia and pulse-rate usually return to normal after the operation and the condition of the patient appears completely satisfactory; in Patterson's case he was even sent home. However, as the new infection gradually sets in, the temperature and pulse begin to rise again. The infection starts as soreness, redness, and œdema about some small part of the wound, or more commonly about the stitch-holes, particularly of the tension sutures. The process gradually spreads, and in the second or third week necrosis takes place in the centre, with formation of black leathery sloughs which then separate and leave a relatively healthy base covered with red granulation tissue. In no single case were the muscles or any of the deeper tissues affected, and the original wound or sinus in most cases healed up; or if it continued to drain, as in most of the thoracotomy cases, the sinus itself looked very healthy.

In places the edge shows a tendency to heal and becomes shelving and non-œdematous, and epithelium grows out across the base to meet small islands of deep epithelium arising from sweat glands or hair follicles that have not been destroyed. The remainder of the edges are raised, undermined, and œdematous, and spread slowly[6] (about 4 mm. a day) but relentlessly, and unless checked by surgical measures enormous areas of the skin of the body are ulcerated. The most extensive lesions were seen in the cases following empyemata; in Poate's case[21] the whole back from the occiput to the middle of the buttocks on both sides was involved. In several of the abdominal cases the entire abdomen was affected, while in addition in one case[6] it extended to involve as well the whole anterior and lateral aspects of the thigh down to the knee, and in another[9] the whole area between the right scapular spine and the iliac crest. A marked feature is the exquisite tenderness of the active edges, which leads to great difficulty with dressings and in more than one case even required general anæsthesia for these to be carried out. This great pain on the slightest manipulation was a feature on which particular stress was laid in twelve of the cases.

Most cases were accompanied by an irregular temperature up to 99° or 100° and sometimes 101° in the evening (102° and 103° in the later stages in our case) and a raised pulse-rate averaging 100 to 120. Most patients suffered from exhaustion from pain and lack of sleep rather than toxæmia, and several records state that the appetite remained good throughout. A moderate leucocytosis was found in some cases. A full post-mortem on Poate's case[21] showed no abnormalities of any viscus other than those usually associated with chronic empyemata. Microscopic study of the edge of the lesion[23] shows extensive fragmentation of the dense subcuticular connective tissue and a heavy cellular infiltration of the subcutaneous fat. There is no thrombosis of the vessels, which are universally dilated and filled with blood with a large number of polymorphs clinging to the walls. This suggests that the gangrene is due to the direct action of some lytic substance on the tissues rather than a cutting off of the blood-supply. Masses of Gram-positive cocci are seen in the centre of the lesion, and scattered organisms in diploform or short chains in the periphery.

DIFFERENTIAL DIAGNOSIS

Before discussing the etiology of this interesting condition it is important, in order to avoid confusion, to differentiate and discriminate between "progressive gangrene" of the skin and other similar conditions:—

1. Common wound infection which may go on to abscess formation and cellulitis and will respond usually to opening up the tissues. If the organism is particularly virulent or there is general or local lowering of resistance on the part of the patient, sloughing of the tissues and acute gangrene of the abdominal wall may occur as in a case fully described recently by Horsley.[40] Before the days of aseptic surgery infectious gangrene was a frequent post-operative complication, and vivid descriptions of its horrors may be found in many of the older text-books.

2. Erysipelas, which, though it may resemble it on the first day, never leads to progressive ulceration.

3. Gas gangrene, with its much more severe symptoms, the prostration of the patient, the foul-smelling discharge, with crepitation of the surrounding œdematous, discoloured skin, and the positive bacterial findings of anaerobic gas bacilli. Recently a case confined to the skin and subcutaneous tissues has been described.[50]

4. "Hæmolytic streptococcal gangrene" of the skin. Meleney gives an excellent description with photographs of a series of 20 cases in China,[41] and later of 11 cases in New York,[46] of hæmolytic streptococcal gangrene affecting the skin and superficial tissues only, occurring chiefly on the limbs after a small injury or apparently spontaneously, the lesions frequently being multiple. The onset is acute, the patient prostrate, often with a positive blood culture. Hæmolytic streptococci are the only organisms invariably present. A better known subgroup of this variety, spontaneous hæmolytic streptococcal gangrene of the scrotum, has been recorded by several authors, Campbell's contribution[42] with five of his own cases being a good example.

5. "Ecthyma gangrenosum" occurring in very young children in a poor state of health and frequently following a predisposing illness, usually varicella. The lesions are often multiple and may be extensive. Ecthyma gangrenosum may occur as a wound infection in debilitated children, especially where there has been contamination with urine or fæces. Lately several isolated cases, usually with multiple lesions, have been described in adults, and recently a more chronic form of pyodermia has been receiving a good deal of attention from German observers. A full list of references to these cases has been given by Brunsting,[22] who described four cases of his own, one with extensive gangrene of the abdominal wall, in patients greatly debilitated with ulcerative colitis. Ecthyma gangrenosum is usually attributed to hæmolytic streptococci, and most authors regard it as a more extensive and deeper form of impetigo.

6. Infection with specific organisms: (*a*) Diphtheritic bacilli, as in the case of Smith,[43] where a spreading ulceration of the abdominal wall followed appendicectomy; diphtheritic bacilli being isolated from the lesion, which responded well to anti-diphtheritic serum. (*b*) Blastomycotic and actinomycotic organisms. (*c*) Tubercle bacilli. (*d*) Leishman-Donovan bodies, as in oriental sores. (*e*) The lymphogranulomatous ultra-microscopic virus which causes intractable ulcers when the buboes break down.

7. "Amœbiasis cutis" may give a somewhat similar picture, as can be seen

by the descriptions of Heimburger,[45] who reviewed ten cases, most of which followed rupture of amœbic abscesses. He found that they responded promptly to emetine. Cole and Heideman[17] describe a case of superficial gangrene following drainage of an appendix abscess with appearances identical with many in this series, in which amœbæ were consistently present in the pus exuded from the ulcerating edges, though there was no history of dysentery and examination of the appendix showed no amœbæ. The spread of the process was definitely checked by emetine hydrochloride intravenously, but there was relapse as soon as the drug was stopped. Eventually it was necessary to excise the margins, after which satisfactory healing promptly occurred. The features were so similar to other cases described in this series that they suggested that " progressive superficial gangrene " was due to infection with amœbæ. The failure of emetine to cure the condition and the final necessity of excision suggests that this particular case was not pure amœbiasis cutis. In no other cases have these organisms been discovered, and Brunsting[22] and Meleney[23] and others, having read this suggestion, have had their cases examined by special experts, with negative results.

8. Foul sloughing infections may occur in wounds produced by human bites and where human saliva appears to have infected the deeper parts of the wound. The gangrene involves the deep tissues, and multiple sinuses are produced. The infecting organisms are fusiform bacilli and spirochætes. Flick described five cases and has reviewed the literature.[51]

ETIOLOGY

In none of the reported cases have any specific organisms been found except amœbæ in the case already discussed. In the majority of cases where full reports are given repeated special examinations have been made to exclude diphtheria, tuberculosis, blastomycosis, etc. In smears and cultures from the discharging pus a variety of organisms have been described. Streptococci were found in all 19 instances where bacteriological reports are included, 14 cases being non-hæmolytic. Staphylococci were found in 11 cases; diphtheroid and Gram-negative bacilli were frequently present.

In none of the cases was there evidence of any underlying disease. In no case was glycosuria reported, and the Wassermann reaction was always negative. In view of the not infrequent chronic ulcers found on the legs of patients with acholuric jaundice,[39] it is interesting to note that in our case three other members of the family were suffering from acholuric jaundice. Our patient, however, showed no evidence of the disease beyond a very slight increase in the fragility of the red cells. Lynn[24] was impressed by the fact that all the cases occurred in patients who required prolonged drainage and restricted diet and were consequently in a run-down general condition. He thought it significant that 50 per cent of the cases occurred after the age of 45. But many of the cases occurred in previously healthy people after an acute infection of only a few days, and a third of the cases occurred in the prime of life below 40. Lowering of the general resistance no doubt assists in the establishment of the infection, but it is not of fundamental importance.

There is no evidence to support as general etiological factors the suggestions of Borelli,[3] who blamed the use of tincture of iodine in his case, and of Tennant,[20]

who, having seen the two cases[19, 20] in which cholecystitis had previously been present, thought they were due to hepatic insufficiency.

With the observations of Arthus and others it is well established that frequent cutaneous necrosis due to hypersensitivity occurs in rabbits and guinea-pigs that have repeatedly been used for injections of bacterial and protein products. Necrosis of the skin at the point of injection has been observed clinically in human beings after diphtheria antitoxin and scarlet fever serum by Gatewood and Baldridge[47] and by others since. One dramatic case[46] is described of a girl who, having received seven weeks previously scarlet fever serum prophylactically, was given a large dose of the serum into the buttock four days after developing scarlet fever. A rapidly-spreading acute superficial gangrene followed over the whole of the buttock, thigh, and abdomen, which was cured by excision and skin-grafts. It was believed that the gangrene was due to the second injection being given while the patient was in a hypersensitive state. Ballin and Moore come to the conclusion that their cases[27 29] of post-operative progressive superficial gangrene are examples of the Arthus phenomenon. They then describe a series of inoculation experiments into the skin of rabbits of a mixed culture of streptococci and diphtheroid bacilli obtained from one of their cases. In one rabbit re-injection with a large dose after a sensitization period resulted in an ulcer with undermined edges and a central slough. Hypersensitivity may be a factor in the production of these post-operative cases, but until further proof is forthcoming it cannot yet be used to explain the whole process. It may be pointed out that Ballin and Moore had only one positive experiment, and none of the clinical cases following serum injections have completely resembled the slow progressive gangrene which is so characteristic.

The most important contribution towards the understanding of the etiology comes from Meleney,[4] who in 1926 was able to grow from the excised lesion a micro-aerophilic streptococcus in cultures taken from regions outside the œdematous margin, and streptococci and staphylococci in cultures from the sloughs, the streptococcus appearing to prepare the way for the actual production of the gangrene by the staphylococcus. These organisms inoculated together into guinea-pig or rabbit's skin invariably produced a gangrenous ulcer, while either of them injected separately failed to produce gangrene. Later in 1931 he was able to confirm these findings on a second typical case.[23] The same micro-aerophilic streptococcus was present in the periphery of the lesion, and again a lesion could only be produced by injecting both organisms together after mixing the cultures. Further, he showed that the association must be extremely close, as if injected separately, even in the closest juxtaposition or after an interval, no ulceration occurred. He found that the staphylococcus was not specific, as certain other staphylococci would produce a similar but not so marked lesion. He concluded that progressive gangrene is caused by synergistic activity of a symbiosis of a micro-aerophilic streptococcus and a staphylococcus. This streptococcus belongs to a group called by Prevot[49] *Streptococcus evolutus*, as they are anaerobic on first culture but after a number of plantations can grow aerobically. This organism is frequently found in the human intestine and in peritoneal exudates, and Prevot found them in the pus of all cases in a series of lung abscesses and bronchiectatic cavities. It is significant that 34 of the 37 cases of progressive gangrene have occurred after operations involving contamination with intestinal organisms or after empyemata. In 1933 Meleney had the opportunity of examining bacteriologically three further cases and was

again able to demonstrate the same streptococcus in each case. It is highly important that further bacteriological experiments on these lines should be carried out by those who may meet these rare cases in the future.

The infection has appeared to start round tension sutures in many of the cases and special emphasis has been placed on this by several authors,[3, 13, 17, 23] and Patterson[30] even suggests that as a prophylactic measure tension sutures should never be used in cases of abdominal and chest abscesses. Tension sutures would therefore appear to be a factor in establishing the infection.

TREATMENT

A study of the results of treatment leaves no doubt as to the correct therapeutic procedure. Non-operative measures have all proved unavailing. These have included high vitamin diets, analgesics, and sedatives for sleep, all varieties of local applications, antiseptic and hypertonic; heat, X-rays, direct and artificial sunlight, which usually lead to irritation; metallic injections of arsenic, antimony, and manganese; antisera, vaccines, staphylococcal toxoid, protein shock, diphtheritic antitoxin, and repeated blood transfusions. In the 37 cases there appear to be only 3 exceptions; one after two months responded to quartz light,[16] one to 6 per cent sodium chloride after involving the entire abdomen and both flanks,[11] and credit was given to general hygienic measures and the use of immunized blood transfusions in the third case, which had failed to respond to repeated attempts at excision.[18] Of the others not treated surgically, 3 of the post-empyemata cases died after many months, the greater part of the trunk being involved;[21, 22, 36a] one died of nephritis before excision could be carried out;[20] having refused excision, another recovered after ten months of pain and suffering when the lesion "burnt itself out";[12] the remaining one healed spontaneously after four months of such intolerable pain that the patient's morale was undermined and suicide was committed shortly afterwards.[20]

All cases in which radical measures were adopted recovered with two exceptions, one a man of 70 who died of pneumonia[26] and another who died of endocarditis,[31] the spread in both cases having been arrested. An incision with a cautery knife should be made beyond the advancing margin through the healthy skin and subcutaneous tissue down to the muscular layer so as to circumscribe the lesion. Excision of the entire ulcerating edge is then carried out. If a scalpel is used in extensive lesions as in two cases,[1, 15] the tissues must then be coagulated in order to check the hæmorrhage, which may be so considerable as to render blood transfusions necessary.[2] If the lesion is extensive, the excision may have to be done in several stages, as in Christopher's case.[2] It was only necessary to cauterize the base in one case.[15] The results were almost without exception immediate and gratifying, a pleasing feature being the instant cessation of the severe pain. Inadequate excision, however, was usually followed by prompt recurrence necessitating a repeat of the operation.[4, 14, 18, 21, 30] Thiersch or pinch skin-grafts (used in 7 cases) may be applied after a short interval to assist healing, but the lesions usually heal spontaneously and sufficiently rapidly, especially where islands of new epithelium are present in the centre round the original wound. Should the gangrenous ulcer already be so far advanced that complete excision of the edges appears too formidable an operation, a barrier may be made in the healthy skin

beyond the œdematous margin by circumscribing the lesion with an incision down to the muscle and perhaps packing with 1 per cent formalin.[4] In all three cases[4, 10, 32] where this procedure was adopted the spread was arrested and satisfactory healing resulted.

It is hoped that in future this type of gangrene will be recognized during the first few weeks and the whole lesion immediately excised. The scalpel should then be used for preference, as no time is lost while the tissue destroyed by the cautery is separating.

SUMMARY

1. A case of a rare complication suitably called "progressive post-operative gangrene of the skin" is described. The importance of using a definite nomenclature to facilitate recognition and reference is emphasized.

2. Only one other case has been recorded in England, but 37 cases have been collected from the foreign literature and reviewed. Their similarity justifies grouping them together as an entity.

3. The rarity of the condition is shown by an analysis of all cases of post-operative gangrene of the abdominal and chest wall occurring in the London Hospital from 1903–33. One case only may be an example of "progressive post-operative gangrene of the skin", but the evidence available is too scanty to include it in the series.

4. The most characteristic features are: Its occurrence usually after drainage of an appendix abscess or empyema; the slow but steadily progressive spread; the involvement of superficial tissues only; the raised, œdematous, undermined and exquisitely tender edges; the relatively healthy base of red granulation tissue; the healing of the original sinus; the extensive area involved unless checked; and its failure to respond to any non-operative therapeutic measures.

5. Treatment consists of early recognition and excision of the entire lesion; or if first seen in the later stages, of excision of the advancing edges with the cautery. The results are immediate and gratifying.

6. Bacteriological experiments suggest that it is due to the synergistic activity of a symbiosis of a micro-aerophilic streptococcus present in the pleural or peritoneal exudate and a non-specific staphylococcus introduced from without. Tension sutures appear to play a part in the establishment of the infection. Cutaneous hypersensitivity (similar to the Arthus phenomenon) may be an important etiological factor.

7. The condition is little known in this country. Failure to recognize it may lead to failure to carry out the only effective treatment, with consequent death of the patient or months or even years of pain and suffering.

I wish to express my gratitude to Dr. Hutchison and Dr. Rowlands for their kind permission to publish this case.

REFERENCES

Case References—

[1] CULLEN, T. A., *Surg. Gynecol. and Obst.*, 1924, xxxviii, 579.
[2] CHRISTOPHER, F., *Surg. Clin. N. Amer.*, 1924, iv, 795.
[3] BORELLI, C., *Giorn. Ital. d. Mal. Ven.*, 1924, lxv, 326.

[4, 5, 6] BREWER, G. E., and MELENEY, F. L., *Ann. of Surg.*, 1926, lxxxiv, 438.
[7, 8, 9] PORTER, C. A., *Trans. Amer. Surg. Assoc.*, 1926, xliv, 408.
[10] MOSCHOVITCH, *Ibid.*
[11] CLINTON, *Ibid.*
[12] ALEXANDER, E. G., *Ann. of Surg.*, 1926, lxxxiv, 461.
[13] MAYEDA, T., *Deut. Zeits. f. Chir.*, 1926, cxcix, 350.
[14] SHIPLEY, A. N., *Ann. of Surg.*, 1928, lxxxvii, 245.
[15] GILLESPIE, M. G., *Ibid.*, lxxxviii, 248.
[16] GORDON, *U.S. Vet. Bur. Bull.*, 1928, iv, 1045.
[17] COLE, W. R., and HEIDEMAN, M. L., *Jour. Amer. Med. Assoc.*, 1929, xcii, 537.
[18] PROBSTEIN, J. G., and SEELIG, M. G., *Surg. Gynecol. and Obst.*, 1928, xlvii, 247.
[19] FREEMAN, L., *Ann. of Surg.*, 1930, xcii, 779.
[20] TENNANT, *Ibid.*, 783.
[21] POATE, R. G., *Med. Jour. of Australia*, 1930, ii, 398.
[22] BRUNSTING, L. A., *Arch. Dermatol. and Syph.*, 1930, xxii, 655.
[23] MELENEY, F. L., *Ann. of Surg.*, 1931, xciv, 961.
[24] LYNN, F. S., *Jour. Amer. Med. Assoc.*, 1931, xcvii, 1597.
[25] WARFIELD, L. M., *Ann. Clin. Med.*, 1927, v, 884.
[26] MITCHELL, J. F., Read before Baltimore City Medical Society, 1930, Dec. 5. Quoted by Lynn.
[27, 28, 29] BALLIN, M., and MORSE, P. F., *Amer. Jour. Surg.*, 1931, xi, 81.
[30] PATTERSON, A. P., *Ann. of Surg.*, 1932, xcvi, 1091.
[31] BAKER, W. H., and TERRY, C. C., *Jour. Amer. Med. Assoc.*, 1932, xcviii, 138.
[32] SCOTSON, F. H., *Lancet*, 1933, i, 80.
[33, 34, 35] MELENEY, F. L., *Surg. Gynecol. and Obst.*, 1933, lvi, 847.
[36] CAROL, W. L., *Nederl. Tijds. v. Geneesk.*, 1932, lxxvi, 1838.
[36a] STEWART-WALLACE, A. M. Reported above.

Other References—

[37] MELENEY, F. L., *Ann. of Surg.*, 1930, xci, 289.
[38] MCDONAGH, J., *Lancet*, 1933, i, 163.
[39] VAUGHAN, J., *The Anæmias*, 1934. London.
[40] HORSLEY, J. S., *Arch. of Surg.*, 1929, xix, 882.
[41] MELENEY, F. L., *Ibid.*, 1924, ix, 317.
[42] CAMPBELL, M. F., *Surg. Gynecol. and Obst.*, 1922, xxxiv, 780.
[43] SMITH, S. W., *Brit. Med. Jour.*, 1931, ii, 800.
[44] STANNUS, H. S., *Proc. Roy. Soc. Med.*, 1932, Nov.
[45] HEIMBURGER, L. F., *Arch. Dermatol. and Syph.*, 1925, xi, 49.
[46] MELENEY, F. L., *Ann. of Surg.*, 1930, xci, 289.
[47] GATEWOOD and BALDRIDGE, *Jour. Amer. Med. Assoc.*, 1927, lxxxviii, 1068.
[48] MELENEY, F. L., *Ibid.*, 1929, xcii, 2009.
[49] PREVOT, A. R., *Ann. de l'Inst. Pasteur*, 1925, xxxiv, 417.
[50] MOORE WHITE, M., *Brit. Med. Jour.*, 1934, ii, 932.
[51] FLICK, J. B., *Ann. of Surg.*, 1932, xcvi, 118.

ADENOMA OF THE DUODENUM

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EVERY writer on the subject emphasizes the comparative rarity of benign tumours of the gastro-intestinal tract. When the duodenum alone is considered the rarity is greatly enhanced. A review of the literature reveals the following important references :—

Smoler[1] refers to an instance of adenoma of the duodenum recorded by Cruveilhier[2] in 1835. Rokitansky,[3] in 1861, when discussing connective-tissue tumours of the intestine, stated that non-malignant tumours of the duodenum may press upon the bile and pancreatic ducts causing a retention of their contents, but he gave no specific case reports. Salvioli,[4] in 1876, reported an adenoma of Brunner's glands. Perry,[5] in 1893, described a case of papilloma arising in the neighbourhood of the duodenal papilla and causing jaundice ; microscopically it proved to be an adenoma composed of mucous glands. Thompson,[6] in 1897, showed a specimen of polypoid growth of the duodenum at a meeting of the Pathological Society of Manchester ; microscopically the tumour was a fibro-adenoma. Funkenstein,[7] in 1904, reported multiple mucous-cell adenomata in the alimentary tract of a 24-year-old woman, six of which were in the duodenum. Doering,[8] in 1907, noted an instance of diffuse polyposis of the intestines, several of these polypi being in the duodenum, and although the patient—a man—was only 28 years of age, the condition was associated with carcinoma of the transverse colon. The same author collected 50 cases of polyposis of the gastro-intestinal tract, but in 3 only was the duodenum involved. In 1908 Versé[9] reported a case in which two adenomata were found in the duodenum. In 1910 Wechselmann[10] referred to an instance of mucous-cell polyposis of the intestinal tract in which the duodenum was involved. In 1916 Weishaupt[11] described a duodenal tumour consisting of Brunner's glands with a fibromuscular stroma in a child 11 days old. Willis and Lasersohn,[12] in 1925, reported two instances of adenomata of the duodenum. In 1928 Golden[13] added two cases to the list : one of adenoma of Brunner's glands and the other a mucous-cell adenoma which could be partially pushed back through the pylorus. This worker brought the total of non-malignant tumours of the duodenum up to 19, but this list included myoma, hæmangioma, papilloma, and others outside the adenomatous group. In 1929 Balfour and Henderson[14] reported 6 cases of benign tumours of the duodenum, 3 of which were adenomata. They refer to the fact that in 119 cases of benign tumour of the intestine recorded by King, the duodenum was involved in only 5 instances, again illustrating the rarity of benign tumours in this situation. In 1933 La Roque and Shiflett[15] reviewed the literature to date, but added little to the foregoing as far as benign tumours are concerned.

The description of the microscopic appearances of one of the adenomata reported in the literature (Willis and Lasersohn,[12] p. 955) suggests that this tumour was really a fibroma covered by normal mucous membrane, and there may be a few others included in this group which are not true adenomata. It will therefore be realized that a Brunner adenoma is a rare pathological entity, and for this reason we present the following case.

CASE REPORT

G. B., a man aged 54 years, was admitted to hospital on April 23, 1934. He had had attacks of epigastric pain for the past nine months. The pain occurred about eight hours after food and was relieved by vomiting. During the week before admission he had vomited a considerable quantity of coffee-ground material and had had melæna.

FIG. 445.—Adenoma of duodenum. Pyloric end of the stomach and first part of the duodenum.

Examination of the abdomen revealed tenderness on deep palpation over the epigastrium, but no tumour could be felt. A test-meal showed a rather high gastric acidity and a positive benzidine test on the fasting specimen, and a barium meal demonstrated a penetrating ulcer high up on the lesser curve of the stomach. The patient was examined at the request of Dr. A. V. Campbell, and it was decided that as the ulcer was a simple one medical treatment should be continued.

He was treated by diet, alkalis, and belladonna and discharged from hospital on June 11, with no symptoms and having gained 7 lb. in weight.

The symptoms, however, soon recurred and he was re-admitted on Sept. 1. A further X-ray was taken and showed the same ulcer and a duodenal cap which was normal in outline

and filled well. Surgical treatment was then deemed advisable. However, at this time the patient's general condition was poor, as he had an obvious secondary anæmia and a bronchitis with some dullness over the right chest. The bronchitis improved with treatment and he was given a blood transfusion (16 oz.) two days before operation.

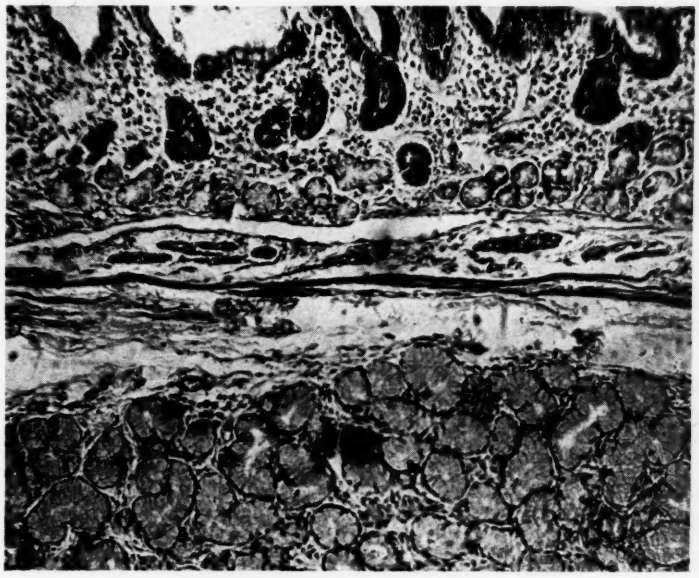

FIG. 446.—Adenoma of duodenum. (× 140.)

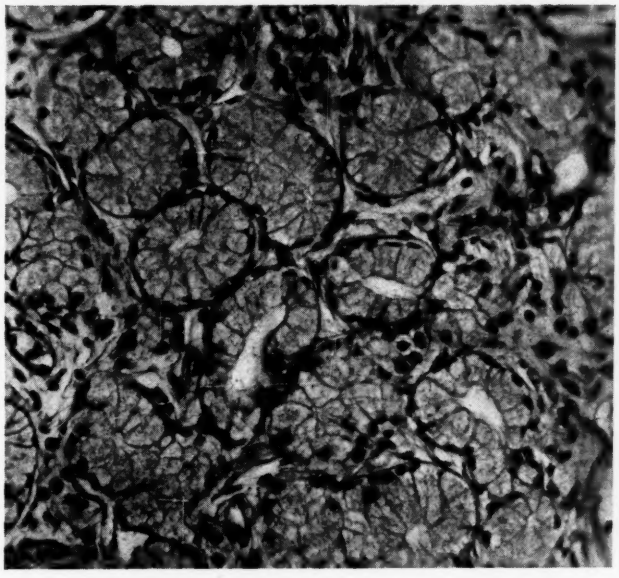

FIG. 447.—Adenoma of duodenum. (× 400.)

OPERATION (Sept. 24).—The anæsthesia used was intratracheal gas and oxygen and local infiltration of the abdominal wall with 1 per cent novocain. The abdomen was opened through a right paramedian incision and the ulcer found rather high up on the lesser curvature.

A partial gastrectomy was decided on, and while palpating the pyloric end of the stomach a tumour was felt in the first part of the duodenum. This was about the size of a small plum and freely movable, obviously pedunculated. The presence of the tumour caused no difficulty in the operation, but rather more of the duodenum than usual was removed. A Billroth I type of anastomosis was performed, and the condition of the patient at the end of the operation was quite satisfactory.

SUBSEQUENT PROGRESS.—Post-operative recovery was uneventful. The patient was allowed up on the twelfth day and was transferred to a convalescent home on the seventeenth day. His doctor reports that since he has returned home he has been quite well.

On reconsideration of this patient's history and examination in view of the operative findings, there does not appear to be anything more than can be fully accounted for by the gastric ulcer, and it seems probable that the duodenal tumour was not giving rise to any signs or symptoms.

PATHOLOGICAL REPORT.—The specimen (*Fig.* 445) consists of the pyloric end of the stomach and the first part of the duodenum. There is a submucous hæmorrhage 1 in. long just proximal to the pylorus. The duodenum contains a pedunculated mass, the pedicle being attached to the posterior duodenal wall about $\frac{3}{4}$ in. distal to the pyloroduodenal junction. The pedicle gradually expands into a pear-shaped tumour of firm consistence and shows slight nodulation. There is no ulceration. Total length of polypus $1\frac{3}{4}$ in.; circumference of pedicle at its narrowest part $\frac{3}{4}$ in.; circumference of tumour at its widest part $3\frac{1}{2}$ in.

Microscopically (*Figs.* 446, 447) there is extensive hyperplasia of the duodenal glands, mainly beneath the muscularis mucosæ, and although these glands show some slight variation in size, their architectural features are well preserved and there are no abnormal mitotic figures. The tissues of the bowel are displaced to accommodate the mass, but there is no infiltration. The stroma is sparse and consists of loose connective tissue containing congested blood-vessels and there are scattered accumulations of small round cells. The mucous membrane covering the tumour is intact.

This microscopic picture closely resembles the description which Golden gave of one of his cases in 1928. He also found accumulations of round cells scattered throughout the tumour which were very similar to those described in the present instance. The condition is well shown in the photomicrographs.

DISCUSSION

Consideration of the recorded cases shows that many of the benign tumours of the duodenum do not cause any clinical symptoms. This is in direct contrast with similar tumours in other parts of the gut, which are so liable to form the starting-point of an intussusception. It is probable that the relative fixation of the duodenum prevents a similar occurrence in this part of the intestine.

While it is true that in one of the reported cases[6] an adenoma growing from the region of the pylorus is stated to have caused partial invagination of that structure into the duodenum, the patient had no symptoms referable to this during life, and it can hardly be classed as an intussusception.

The earlier literature on the subject is chiefly made up of cases found at autopsy, or, since the more frequent resort to laparotomy, of those in which the duodenal tumour has been discovered during the course of an operation for some other disease. In fact, the number of cases in which a duodenal tumour has been the only lesion found, and so may be presumed to be the cause of the symptoms noted, is so small that a true symptomatology is still very difficult.

There does not appear to be any special age incidence, the youngest patient being 11 weeks old and the oldest 75 years. Intestinal obstruction is a rare event, and in the few cases in which it has been recorded the tumour has been very large and palpable through the abdominal wall. As a rule the growth is small, and

although some degree of dilatation may be present, the fluid contents of this part of the gut readily pass, even where the lumen is considerably diminished.

When symptoms are present they are usually epigastric discomfort and perhaps pain of a colicky type, but seldom as severe as that associated with an ulcer. Nausea or vomiting is not uncommon and attacks of diarrhœa have been noted several times. The presence of melæna or occult blood in the stools would appear to be the most constant feature of significance and is the only one likely to be of diagnostic aid in the clinical investigation of the patient. The combination of melæna with epigastric symptoms is most likely to suggest the diagnosis of gastric or duodenal ulcer or gastric carcinoma, and the test-meal, and more especially the X-ray examination, are then the most important guides.

In recent years attention has been more keenly focused on the possibility of X-ray diagnosis, and emphasis has been laid on the advantages of fluoroscopic examination with changes of posture in addition to the routine radiogram, but even with up-to-date technique several cases have defied detection. When diagnosis by Roentgen ray is successful there is usually a filling defect of vacuolation type. Golden attaches considerable importance to the presence or absence of a six-hour gastric retention as a means of distinguishing growths arising in the duodenum from those prolapsing into it from the stomach. He states that "in the presence of a filling defect in the duodenal bulb suggesting non-malignant tumour, a six-hour gastric retention may be considered as evidence in favour of a growth arising in the stomach and prolapsing into the duodenum, and the absence of such a retention is indicative of a growth arising in the duodenum itself." Waters[16] points out that a vacuolated type of filling defect points to adenoma, whereas a multilocular filling defect indicates papilloma.

A considerable proportion of instances of benign tumours of the duodenum is associated with pathological conditions elsewhere in the gastro-intestinal tract, such as multiple polyposis, carcinoma, cholelithiasis, and ulceration. The relation of ulceration to lesions in the duodenal glands deserves careful consideration in view of the recent work by Florey and Harding,[17] whose observations give additional support to the view that the normal secretions of Brunner's glands protect the mucosa from damage by the acid gastric juice by virtue of their mucoid consistence and contained alkali. These observers are of opinion that their findings lend further weight to the suggestion that malfunctioning of these glands may be primarily responsible for duodenal ulcer.

As regards treatment, since the presence of a duodenal tumour is so often only discovered during an operation for some other condition, the surgeon must often be guided by circumstances as to the best treatment to adopt. When the associated lesion is a gastric or duodenal ulcer, a radical removal of the ulcer and the tumour would seem the correct method. There is, however, no recorded case of a simple duodenal tumour becoming malignant, and where the growth is polypoid trans-duodenal resection should suffice. In the case of a sessile growth or one in which the possibility of sarcoma or carcinoma cannot be definitely excluded, the portion of duodenum involved should be excised and an end-to-end anastomosis performed.

Our thanks are due to Dr. T. V. Cooper for supplying some of the references; to Dr. Campbell for his notes on the case; and to Mr. W. H. Fussell for his help in preparing the photomicrographs.

REFERENCES

[1] SMOLER, F., *Beitr. z. klin. Chir.*, 1902, xxxvi, 139.
[2] CRUVEILHIER, J., *Anatomie path. du Corps humain*, 1835–42, xxii, 6, from Smoler.
[3] ROKITANSKY, K., *Lehrbuch der pathologischen Anatomie*, 1861, 3rd ed., 231. Vienna: W. Braunmuller.
[4] SALVIOLI, G., *L'Osservatore, Gaz. del. clin. di Torino*, 1876, xii, 481.
[5] PERRY, E. C., *Trans. Pathol. Soc. Lond.*, 1892–3, xliv, 84.
[6] THOMPSON, P., *Lancet*, 1897, i, 383.
[7] FUNKENSTEIN, O., *Zeits. f. klin. Med.*, 1904, lv, 536.
[8] DOERING, H., *Arch. f. klin. Chir.*, 1907, lxxxiii, 194.
[9] VERSÉ, M., *Arb. a. d. path. Inst. in Leipzig*, 1908, v, 1.
[10] WECHSELMANN, L., *Beitr. z. klin. Chir.*, 1910, lxx, 855.
[11] WEISHAUPT, ELISABETH, *Virchow's Arch.*, 1917, cciii, 24.
[12] WILLIS, A. M., and LASERSOHN, M., *Ann. of Surg.*, 1925, lxxxii, 952.
[13] GOLDEN, R., *Amer. Jour. Roentgenol.*, 1928, xx, Nov., 405.
[14] BALFOUR, D. C., and HENDERSON, E. F., *Ann. of Surg.*, 1929, lxxxix, Jan., 30.
[15] LA ROQUE, G. P., and SHIFLETT, E. L., *Ibid.*, 1933, xcviii, Aug., 178.
[16] WATERS, C. A., *Amer. Jour. Roentgenol.*, 1930, xxiv, Nov., 554.
[17] FLOREY, H. W., and HARDING, H. E., *Jour. Pathol. and Bacteriol.*, 1934, xxxix, Sept., 255.

MEASURED DOSAGE IN THE RADIUM TREATMENT OF CARCINOMA OF THE URINARY BLADDER

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Of late years much has been written on the radium treatment of carcinoma of the urinary bladder, and were the object of this contribution merely to state a case for the radiosensitivity of this particular neoplasm it might well be considered superfluous. But radium therapy is entering at the present time into a new phase, in which the old empiricism is being discarded in favour of a system of physically determined dosage, and this paper will show how such a system can be applied in practice to carcinoma of the bladder in such a way that the surgeon will know, firstly, that his dosage is safe, and secondly, that his dosage is the same for all tumours treated, irrespective of their size.

INTRODUCTION

No single dosage unit for gamma-ray therapy has so far found general acceptance, but the fact that a variety of such units is being pressed upon the radiological world from widely different sources goes to prove how general is the dissatisfaction with older methods. The particular unit adopted in any country or clinic matters little, so long as it is a genuine measure of the amount of energy delivered to the tumour and its bed in any stated case. Of these units, however, the 'roentgen' ('r' unit—gamma radiation) is the one which is probably most widely adopted at the moment and which is likeliest to gain international recognition, and it is here accepted.

Research has been active also along another line—the distribution of radioactive foci in any plan of treatment. The basic requirement is the production of a homogeneous field of irradiation which will include the tumour and its bed. If this can be achieved, elimination is secured, firstly of localized necrosis due to high dosage, and secondly of localized areas of sublethal dosage from which subsequent recrudescence is bound to occur in an otherwise adequately treated zone. It is apparent that two separate but interdependent factors are involved, namely: (1) The total amount of radium required; (2) The number and distribution of the foci of radiation. These two factors are fully discussed in a recent article by Paterson and Parker on the application of "measured dosage" to the problems of radium therapy. The present paper deals with the application of the system there outlined to the special case of carcinoma of the urinary bladder. As will be seen in the following pages, the manner of the application is such as to be quite within the reach even of those who have no bent for considerations of pure physics.

TECHNIQUE OF TREATMENT

Preliminary investigations completed, suprapubic cystotomy is performed, and the bladder explored. The growth having been located, the best possible exposure is obtained. Often the bulk of a tumour, especially if pedunculated, prevents adequate access to its base and the surrounding tissues. In such cases it is recommended that the protuberant and obstructive portions be removed by endothermy merely down to the normal level of the mucosa, and without any attempt at complete treatment in this way. The full extent of the growth having been investigated, it remains now to radiate in the best manner possible.

The difficulties inherent in any method of surface application of radium within the bladder are so great that it cannot be said that any such technique based on scientific principles has yet proceeded beyond the purely experimental stage. For the present, therefore, it is desirable to consider only the interstitial approach to the problem.

The first step is to determine the area to be radiated, since the distribution of radio-active foci depends upon the shape and size of this area. Either a circle or a square may be chosen. As the former, however, is almost universally applicable in the bladder, and as the rules for circular implantations can be reduced to simple tabular form (as is shown later), attention will be confined to this distribution.

From the Appendix it will be seen that implantation of a circular zone demands, according to the size of the circle, one of three distributions: (1) An outer circle only; (2) An outer circle with centre point; (3) An outer circle with inner circle of half the radius of the outer one, and a central point. Now, it must be emphasized that the more numerous the foci the more satisfactorily can such distributions be achieved. If for no other reason, therefore, radon seeds would be the vehicles of choice, as being much smaller than any radium needles commonly in use. Their selection, however, is further indicated because, being more easily inserted than needles even when exposure is not of the best, their use shortens the operative procedure. Moreover, arrangements for subsequent removal are eliminated. Study of the Appendix will show how the required distributions of the seeds should be planned ideally, but the surgeon must find the most practicable way of applying these principles, and a rule-of-thumb method of doing so is set forth in *Tables I–III*. Each table is constructed in the same manner, and shows at a glance just how many seeds are required for a circle of any given size between diameters of 3 and 7 cm., and just how the physical requirements are most fully met. *Table I* gives instructions when seeds of 1·1 mc. initial strength are used, this strength being considered best. *Tables II* and *III* apply to seeds of initial strength equivalent to 1·3 mc. and 1·5 mc. respectively, the latter being considered the maximum strength which can be used with any degree of safety. Such seeds, of filtration equivalent to 0·5 mm. gold, have been found satisfactory in practice.

The seeds are implanted at a depth of ½ cm. below the mucosal surface, and the distributions given in the tables are such as to produce a dose of 6000 'r' units on each surface of a disc of bladder wall corresponding to the area treated and of a thickness of 1 cm. Some modification of this dosage level may be found desirable in the light of further experience, but present indications are that it is sufficient, without being dangerous to such healthy tissues as may lie within the disc in question. Every case should be implanted wide of the tumour in every

direction, a circle being selected of such size as to include the whole growth with a margin of healthy tissues around it. Accurate implantation is insisted upon, within the limits of practicability, and the number of seeds required for any circle is disposed at equal intervals around the circumference.

Table I.—Distribution of Radon Seeds of Initial Strength 1·1 mc.

	Diameter in Centimetres of the Various Circular Areas				
	3	4	5	6	7
Total seeds required	8	11	14	19	24
Number in outer circle ..	7	9	10	13	16
Number in inner circle ..	0	0	4	5	7
Centre point	1	2	0	1	1

Table II.—Distribution of Radon Seeds of Initial Strength 1·3 mc.

	Diameter in Centimetres of the Various Circular Areas				
	3	4	5	6	7
Total seeds required	7	9	12	16	21
Number in outer circle ..	6	7	8	11	14
Number in inner circle ..	0	0	4	5	6
Centre point	1	2	0	0	1

Table III.—Distribution of Radon Seeds of Initial Strength 1·5 mc.

	Diameter in Centimetres of the Various Circular Areas				
	3	4	5	6	7
Total seeds required	6	8	11	14	18
Number in outer circle ..	5	6	8	10	12
Number in inner circle ..	0	0	3	4	5
Centre point	1	2	0	0	1

A brief example is appended in order to demonstrate the procedure. Suppose we have a lesion of diameter 4 cm., such that a circle of 7 cm. diameter be found adequate, and that seeds of 1·1 mc. are available. It will be seen from *Table I* that twenty-four seeds will be required: 16 of these should be distributed equally around the circumference of a circle of 7 cm. diameter, 7 around the circumference of a circle of 3·5 cm. diameter, and 1 should be placed at the centre point. All should be approximately ½ cm. deep to the mucosa.

If seeds are not available, recourse must be had to radium needles. These have undoubted disadvantages. They take much longer to insert, and require subsequent removal. In addition, there is a greater liability to accidental injury by penetration of the bladder wall at the time of implantation. Scientific dosage according to modern standards is quite possible of achievement, but obviously the circular method is hardly possible. Some form of square implantation is indicated. The calculation is not difficult, but cannot be reduced to simple tabular form. Full details are available in the article by Paterson and Parker previously referred to.

FIG. 448.—Flexible rule (Dr. Herd). (Manufactured by Messrs. C. F. Thackray, Leeds.)

The taking of measurements within the bladder is not easy, but is facilitated by the ingenious flexible rule devised by Dr. Herd, and shown in *Fig.* 448. *Fig.* 449 shows a useful seed-introducer with handle set at right angles to the barrel. This arrangement obviates obstruction of the view by the hand. The point of the introducer is smeared with sterile vaseline, and the instrument is loaded from the distal end, the vaseline causing the seed to remain in the barrel even in the dependent

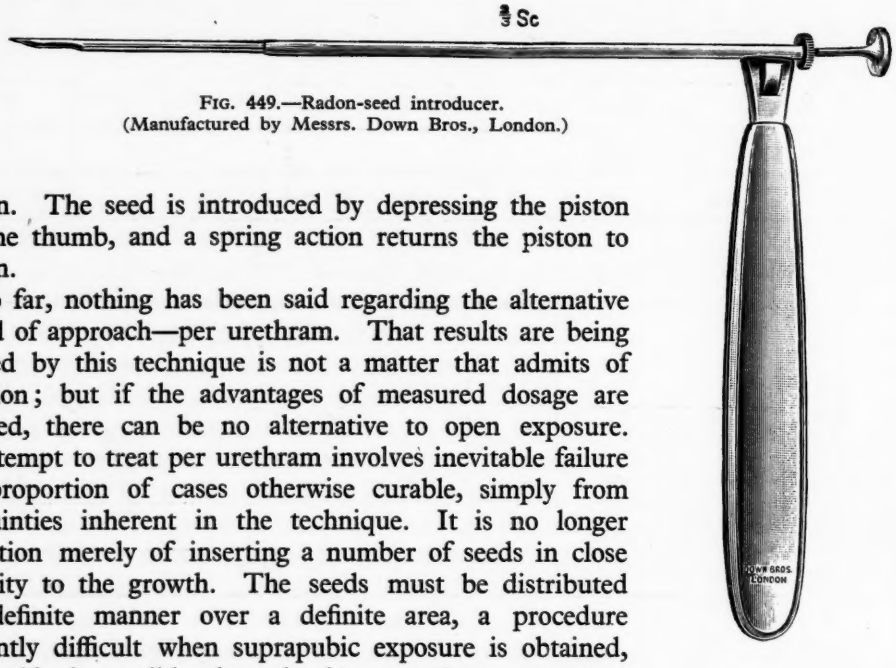

FIG. 449.—Radon-seed introducer.
(Manufactured by Messrs. Down Bros., London.)

position. The seed is introduced by depressing the piston with the thumb, and a spring action returns the piston to position.

So far, nothing has been said regarding the alternative method of approach—per urethram. That results are being obtained by this technique is not a matter that admits of refutation; but if the advantages of measured dosage are conceded, there can be no alternative to open exposure. Any attempt to treat per urethram involves inevitable failure in a proportion of cases otherwise curable, simply from uncertainties inherent in the technique. It is no longer a question merely of inserting a number of seeds in close proximity to the growth. The seeds must be distributed in a definite manner over a definite area, a procedure sufficiently difficult when suprapubic exposure is obtained, and frankly impossible through the operating cystoscope. In short, radium implantation by the per-urethral route is a procedure which must be universally condemned when cancer is suspected. In support of this, the writer would quote Barringer in the statement that "even the smallest bladder tumour, if it be cancerous, is a grave disease not to be treated by inadequate means."

RESULTS OF RADIUM TREATMENT

In conclusion it must be emphasized that the omission of the consideration of purely surgical methods of dealing with carcinoma of the bladder is not an indication that these are regarded as being unimportant. Time alone will show which cases are best handled surgically and which best dealt with by radium. *Table IV* shows the results of treatment in the Radium Institute, Manchester, for 1931.

Table IV.—Results of Treatment of Carcinoma of the Urinary Bladder (Radium Institute, Manchester, 1931*)

Total cases treated	.. .. ..			9
Alive	.. ..	Well ..	3	4 (44·4 per cent)
		Diseased ..	1	
Dead	.. ..	Post-operative	1	5 (55·5 per cent)
		Malignancy ..	4	

* Interval since treatment, over two and under three years.

This represents the results as achieved in the early days of the present phase when knowledge of radium distribution was much less perfectly worked out than now, and when only inoperable cases were treated. Complete homogeneity of radiation could not be claimed, and yet the figures at a period of over two and under three years show 33·3 per cent of cases alive and well. As indicative of the type of case going to make up this group it may be stated that the smallest growth measured 4 × 3 cm. (in this case a second smaller growth was present in addition), and that the remainder varied from this up to an enormous one measuring 14 × 12 cm. All except one involved the base of the bladder. In such cases most surgeons will prefer radium to extremely radical surgical extirpations with their high mortality and other attendant disadvantages. Differences of opinion must arise, however, in the early cases, and then the best interest of the patient can be served only by due consideration of the individual circumstances of the case by one who is fully alive to the possibilities of both radium and surgery.

SUMMARY

1. Recent advances in radium technique are described in their application to the treatment of carcinoma of the urinary bladder.

2. A concise guide is supplied to the arrangements and amounts required in actual practice, based on the finding that 6000 ' r ' is the satisfactory dose.

3. The question of the surgical approach is discussed, and cystoscopic methods of treatment are condemned.

4. Some results are quoted.

APPENDIX

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In planning a scheme of radium distribution to produce homogeneous and calculable radiation the use of circular arrangements is especially favourable. On the one hand, the circles have a wide range of clinical applicability, and, on the other hand, the difficulties of calculation are reduced to a minimum. The variation

of intensity over a plane at 0·5 cm. from radio-active circles of different diameters is shown in *Fig.* 450. It will be seen that the small circles give uniform irradiation, whereas the larger circles show a progressive weakening of the central intensities as the size increases. It is obvious from the shape of the distribution curves

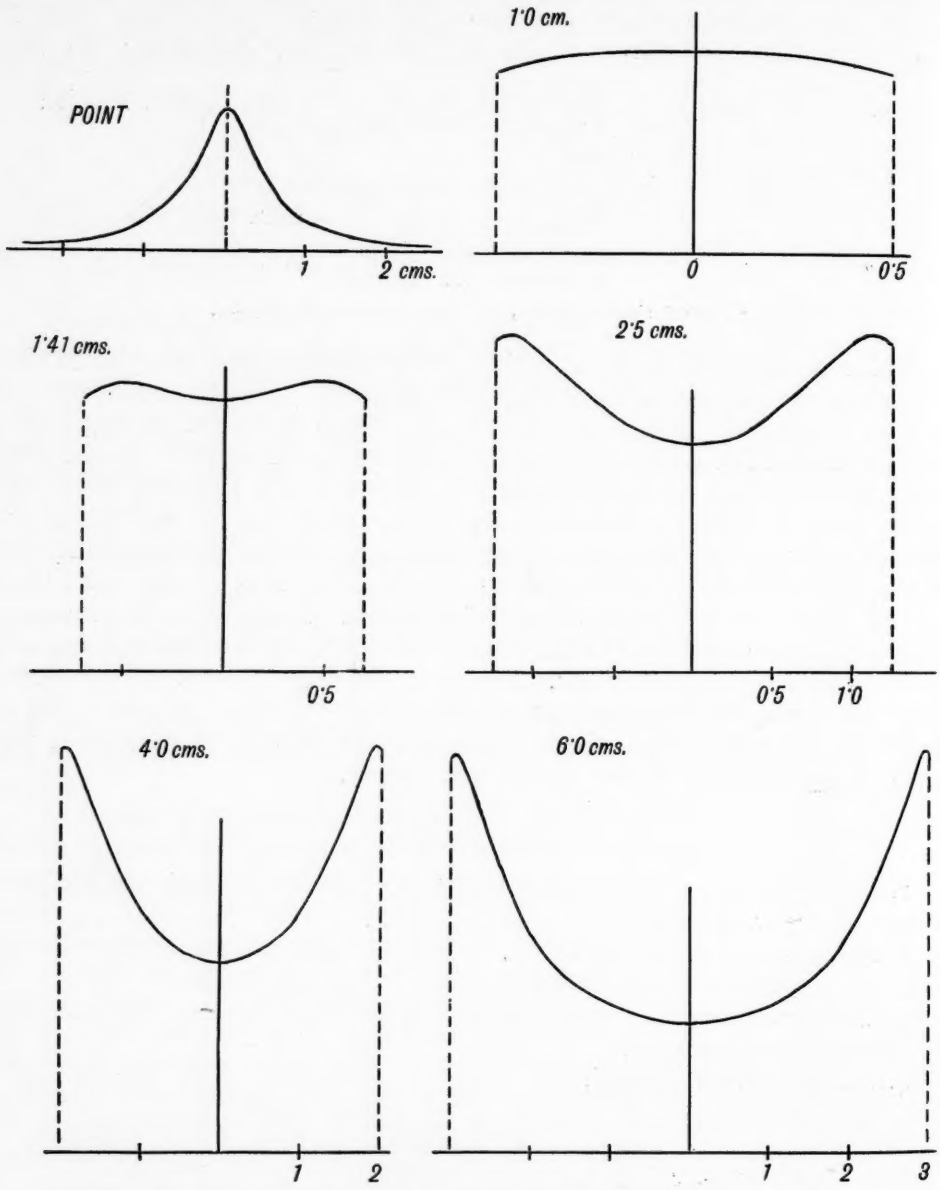
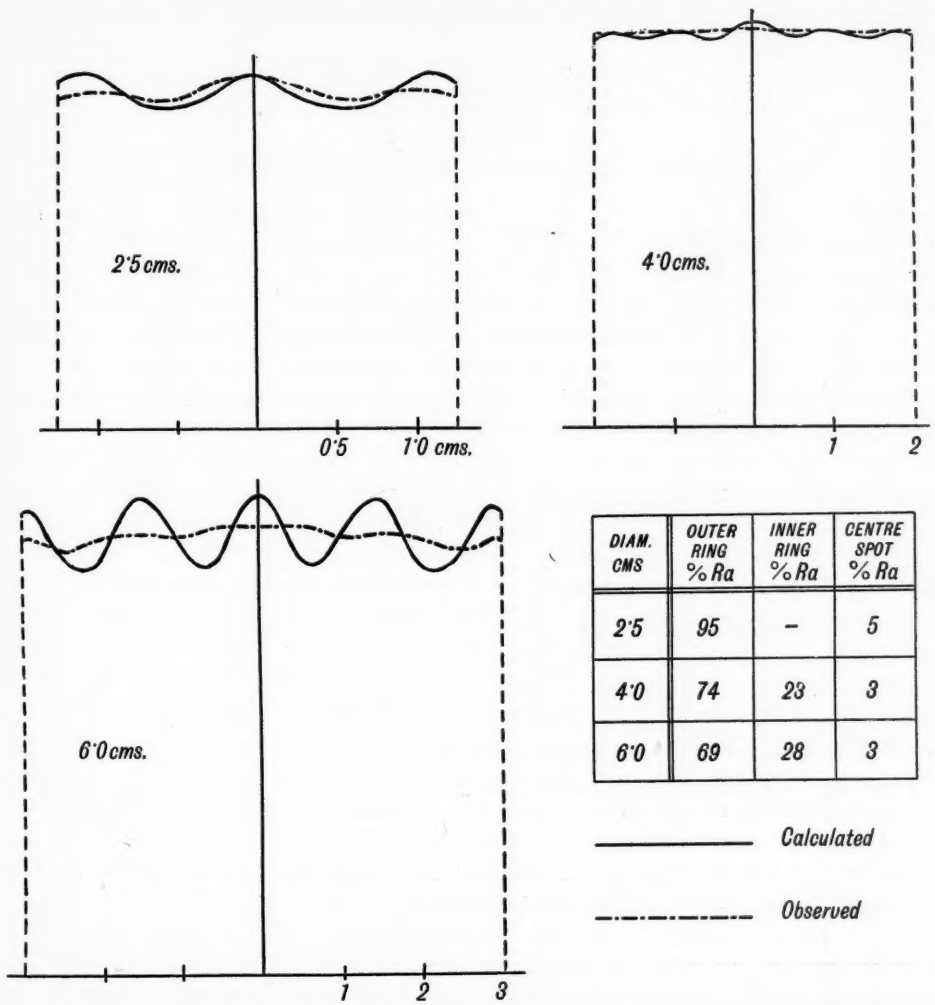


FIG. 450.—Distribution of intensity at ½ cm. from radio-active circles of different diameters. The dotted ordinates define the limits of the treated area.

that for any total diameter it is possible to develop a combination of rings to deliver radiation homogeneous to any required degree. With a permissible variation of ± 10 per cent, a combination system possessing the necessary clinical simplicity

has been developed by R. Paterson and the writer (*Brit. Jour. Radiol*, 1934, Oct.). For diameters between 1·5 and 3 cm. it is sufficient to place 5 per cent of the total radium at the centre. For diameters of 3 or more cm., a centre-spot of 3 per cent and an inner circle of half the total diameter, with a particular percentage of radium, are adequate. The modified distributions of intensity are shown in *Fig.* 451. The



DIAM. CMS	OUTER RING % Ra	INNER RING % Ra	CENTRE SPOT % Ra
2·5	95	–	5
4·0	74	23	3
6·0	69	28	3

FIG. 451.—Calculated and observed distributions of intensity at ½ cm. from three compound circular applicators. The greater degree of homogeneity of the experimental curves is mainly due to the finite size of the micro-ionization chambers. The dotted ordinates define the limits of the treated area.

required percentages can be readily assessed from the accompanying graph (*Fig.* 452).

The arrangements outlined above were developed for superficial application, but they may be applied to interstitial treatment without alteration. There will be two parallel planes 1 cm. apart at which the dosage will be uniform and adjusted to say 6000 ' r '. Then no point between these planes will receive less than 6000 ' r ', but on the other hand the dosage within the disc will not greatly exceed 6000 ' r '

except at local foci round each active source, a departure from homogeneity which is well known to produce no deleterious effect.

Experiments with micro-ionization chambers show that the distribution and dosage in tissue agree very closely with those calculated in air on a simple basis.

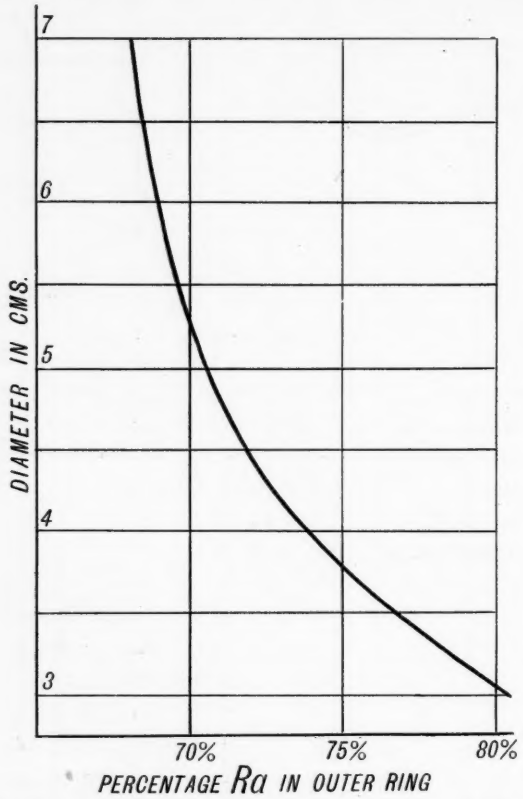


FIG. 452.—Graph showing the percentage of the total radium in the outer circle of the compound applicators: 3 per cent is to be placed at the centre, and the remainder in the inner ring.

Owing to the special type of gamma ray scattering there is no correction factor equivalent to 'back-scatter' in X-ray therapy.

BIBLIOGRAPHY

BARRINGER, B. S., *Surg. Gynecol. and Obst.*, 1934, lviii, May, 867.
PATERSON, R., and PARKER, H. M., *Brit. Jour. Radiol.*, 1934, vii, Oct., 592.
SMITH, A. J. DURDEN, *Brit. Med. Jour.*, 1934, Sept. 29, 584.
SOUTTAR, H. S., *Radium and Cancer. A Monograph*, 1934. London: William Heinemann.
STEVENS, J. THOMPSON, *Radiology*, 1934, xxii, Jan., 99.

INTERINNOMINO-ABDOMINAL (HIND-QUARTER) AMPUTATION

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INTERINNOMINO-ABDOMINAL amputation is fortunately an infrequent operation of surgery, and must remain for all time one of the most colossal mutilations practised on the human frame. Though this mighty ablation was first performed by Billroth[1] in 1891, to Girard[2] belongs the credit of having achieved, in 1895, the first success.

The term 'interinnomino-abdominal amputation' would seem to be the best and most appropriate appellation for this operation, for 'interpelvi-abdominal amputation', the title used by Hogarth Pringle,[3] is rather suggestive of some feat of expert swordsmanship, and would appear to betoken an even more stupendous undertaking than that which is on occasion essayed by surgeons. It is true that in one of our cases a testicle required removal as well, that in another one line of bone section involved the ischial and pubic portions of the opposite innominate bone, and that in one successful case part of the sacrum required removal with the mass; nevertheless, this hyperbolic and ambitious nomenclature hardly seems justified, even although the usual limits of the amputation are transcended by these additional sacrifices of bone or gonad. 'Interilio-abdominal removal' might be applicable to certain more limited growths, but in the cases that have been encountered by the writers nothing but the sacrifice of the whole innominate bone seemed practicable, if the operation were to be effective in its object and purpose. 'Hemipelvectomy' evokes a shudder, and a sigh of regret that some knowledge of Latin and Greek is no longer considered necessary for those who claim to have received more than a kindergarten education.

The literature of the subject has been tabulated by Hogarth Pringle[3] in this JOURNAL in 1916, and by Judin[4] in 1926; the latter author, though writing in English, had been working in Russia, and, perhaps on account of the inaccessibility of references, his table contains a number of inaccuracies. Many of the cases reported in Russian papers and journals are unobtainable in London, and others were personal communications to Judin, so that an accurate scrutiny of these cannot be made. The perusal of the relevant surgical literature indicates, however, that the operation has now been performed at least 79 times by forty-eight different surgeons, but of the reported cases only those of Hogarth Pringle and the present series emanate from British clinics.

The opportunity presented to one of us (G. G.-T.) of performing this amputation as a *single-stage operation* on no fewer than five occasions would seem to be unique. It is true that Hogarth Pringle in his memorable paper already mentioned reported five cases, but in three of these the operation was performed in two stages,

the time intervening between the two séances varying between three months and fifteen months, and in two of the cases the second operation was merely the ablation of a disintegrated os coxæ.

Fortune has kindly ordained that the two authors of this communication have been associated together in the performance of the last four of the five operations that constitute the basis of this paper, and it is perhaps significant that of these four cases only one succumbed. Moreover, the last three cases of the series were operated upon within a few months of each other, and an unparalleled opportunity has thus been afforded us of not only familiarizing ourselves more thoroughly with the technique of the manœuvre, but also of reviewing the anxieties of the undertaking, its technical difficulties and pitfalls, and the post-operative complications. We are furthermore enabled to form some opinion as to the degree of final disablement produced by the ablation and to adjudge the amount of interference with the patient's subsequent *modus vivendi*.

Hogarth Pringle's most admirable paper has been of the utmost assistance to us in the operative treatment of the cases dealt with in this communication, and that author is indeed to be congratulated on his low mortality, when it is remembered that at the time his operations were performed blood transfusion was not readily available, at any rate in Great Britain, and the shock of the operation had to be combated with nothing more serviceable than saline infusion.

The conviction has grown upon us that some description of the operation as we have experienced it might be of value to those who may from time to time find themselves constrained to advise and to perform this amputation, and the sketches illustrating certain of the stages of the undertaking and the anatomical structures displayed in the course of its performance may be an aid to such surgeons as have less experience of this amputation than ourselves.

It is probable that in the future this operation will be less and less frequently performed. Radiation therapy at the moment seems to be displacing the more crude and more maiming methods of ablating cancer, but even with the growing perfection of methods of radiation, the treatment of certain forms of sarcoma may perhaps still occasionally demand this operation, and so long as procrastination remains a characteristic human trait where disease is concerned, and while benign or quasi-benign tumours are allowed to attain colossal and crippling proportions, only this type of procedure will rid the patient of his encumbrance, his pain, and his menace of malignancy.

CASE REPORTS

Case 1.—**Sarcoma of upper end of femur with involvement of innominate bone. Interinnomino-abdominal amputation. Death.**

HISTORY.—A. H., male, aged 25. The patient's father had died of a sarcoma of the leg twenty-three years previously. The patient had complained of pain in the right hip for four years, but there had been no swelling during that period. He sustained a fall on May 16, 1922, after which he became lame. Radiography now revealed the true nature of the malady, and the report from another institution, dated May 17, 1922, was as follows: "Large periosteal sarcoma of the upper end of the right femur, extending from the region of the great trochanter on to the inferior surface of the neck of the bone and for six or seven inches down the shaft. There are also opacities in the acetabulum and ilium above, which may be secondary deposits. There is considerable outward bowing of the shaft of the femur." An X-ray report dated May 22, 1922—a week later—revealed a transverse fracture of the shaft at the lower end of the growth, which had increased in size in that short space of time. The patient was

transferred to my care (G. G.-T.) at Middlesex Hospital as an inoperable case in the middle of June, 1922, and by this time there was radiological evidence of the involvement of the acetabulum by the neoplasm.

On Examination.—The general condition of the patient was poor: he was anæmic; his pulse was 100, and at times irregular; and he suffered untold pain in the region of the right hip and thigh. In the upper and middle thirds of the latter there was a large diffuse swelling; there was marked outward bowing. The right leg and foot were greatly swollen and there was considerable œdema. In the right groin was a hard, fixed mass, extending on to the abdominal wall. The circumference at the upper end of the thigh measured 56 cm. on the right side and only 41 cm. on the left. A diagrammatic sketch of the swelling is appended (*Fig.* 453); the X-rays have unfortunately been destroyed.

It was resolved, if the peritoneum were free of involvement by the new growth, to attempt to eradicate the mass by an interinnomino-abdominal amputation.

Operation (July 7, 1922).—Spinal anæsthesia and general anæsthesia were administered by Mr. A. E. W. Idris. The steps of the operation were those outlined by Hogarth Pringle, but in this case the extent of the growth necessitated the removal of the right testicle as well. Very considerable hæmorrhage was encountered in the extraperitoneal tissues in the neighbourhood of the cut symphysis and the bladder, and despite the fact that the common iliac vessels had been already secured, much blood was lost. Unfortunately all the arrangements for blood transfusion miscarried; the arrangements for transfusion were not organized as they are now, nor were the services of the Blood-Transfusion Society available then. In our emergency we could only avail ourselves of the services of a diminutive "girl-guide", who donated with great spirit, but with little effect! The patient died of shock about six hours after the completion of the operation.

Fig. 453. — *Case* 1. Diagram indicating the extent of growth. The upward extension of the growth on to the lower part of the abdominal wall is shown.

Histological Report (No. 4197/22, dated 26/7/22).—

Macroscopically the tumour had destroyed probably the whole thickness of femur for a distance of about three inches immediately below the trochanter. It had invaded the glands along the external iliac vessels; all except the most recently formed portions contained bone.

Microscopically the section showed the structure of a round-celled sarcoma. The cells showed a tendency to line the small alveoli formed by trabeculæ of fibrous tissue.

Autopsy.—There was one note of encouragement from the autopsy: despite the magnitude of the primary growth there was a complete absence of any metastases throughout the body.

Case 2.—**Osteoclastoma of innominate bone. Interinnomino-abdominal amputation. Recovery.**

History.—W. J., male, aged 17, while playing football sustained a blow in Scarpa's triangle on the left side; the injury was sufficiently severe to lame him and he was unable to continue the game. His incapacity persisted and he consulted his doctor; a radiogram at this period revealed no bony change. For several weeks thereafter the patient was lost sight of by his medical attendant, having betaken himself to some unqualified persons who enjoyed the confidence and patronage of the chief football club of the town in which he dwelt. By these he was massaged and manipulated, but a swelling having now appeared in the adductor region of the left thigh in the vicinity of the cruro-scrotal fold, the patient returned to the doctor whom he had neglected and was promptly sent up to Middlesex Hospital.

On Examination.—A well-marked tumour could now be felt in the upper part of the thigh and in the perineum, where it could be identified on rectal examination and on deep palpation in the urogenital triangle and left ischio-rectal fossa; it was fixed to the pubic bone, was tender on pressure, and was much more considerable on the deep surface of the innominate bone than on its superficial aspect. All movements of the left hip were slightly limited and there was definite wasting of the quadriceps extensor muscle of the affected side.

Radiological Report (Middlesex Hospital).—The left ischio-pubic junction has almost

completely disappeared. There is irregular new bone formation in this region. The remainder of ischium is increased in density and there is a tumour with bone formation encircling it Appearances point to osteosarcoma (*Fig.* 454).

The clinical characters of the tumour, the rapidity of growth, the radiological report, and particularly the marked contrast between the first and second radiograms taken at a short interval of time, together with the history of injury, prompted the diagnosis of a sarcoma of the innominate bone, which was shared by several of my colleagues. The case was considered to be one more likely to be permanently benefited by amputation than by radium therapy; it was thought that the relative inaccessibility of the tumour might tend to imperfect radiation.

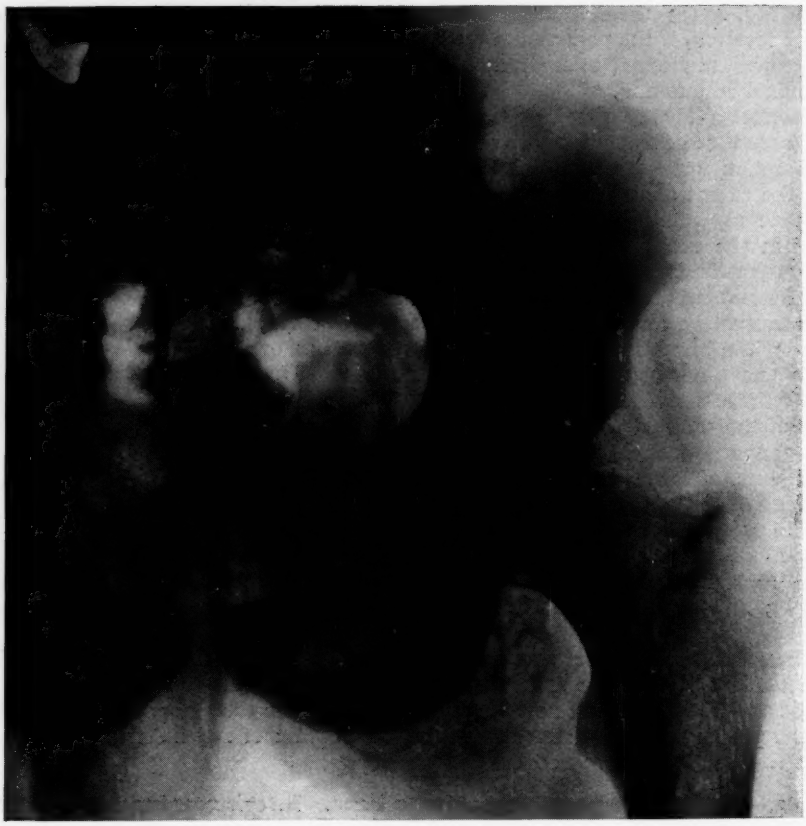

FIG. 454.—*Case* 2. Radiogram of cystic osteoclastoma of left innominate bone. Interilio-abdominal amputation successfully performed, February, 1929.

OPERATION (February 4, 1929).—Interinnomino-abdominal amputation performed by G. Gordon-Taylor. Spinal anæsthesia was induced by Dr. H. P. Crampton, with heavy stovaine; this was supplemented by light open ether. Messrs. Eric Riches, P. Wiles, and H. K. Evans rendered loyal and valuable assistance.

The incision and the steps of the operation were mainly those outlined by Hogarth Pringle. It was deemed important to satisfy oneself at the earliest moment as to the relations of the tumour to the peritoneum, and to convince oneself of the possibility of ablating the neoplasm. The flat muscles of the abdominal wall were therefore cut through at their attachment to the iliac crest at the very commencement of the undertaking. The symphysis required division by sawing. In this case the common iliac vessels were not ligated; the external iliac artery and vein were tied distal to the origin of the deep epigastric vessels. The nerves were injected with novocain and were treated with 80 per cent alcohol after section. The iliac portion of the innominate bone was sawn through lateral to its sacral articulation.

FIG. 455.—*Case* 2. Naked-eye appearance of innominate bone, the seat of a cystic osteoclastoma. Interilio-abdominal amputation successfully performed, February, 1929.

FIG. 456.—*Case* 2. The same bone as *Fig.* 455, showing the growth. View from inner aspect.

Subsequently this articular portion of the innominate bone was found to project unduly, and was easily and expeditiously removed. Immediately before operation 800 c.c. of blood were obtained from the patient's brother, citrated, and kept warm. This was given slowly, commencing immediately the main vessels were divided. A further transfusion of 400 c.c. from another donor was given some hours after the operation.

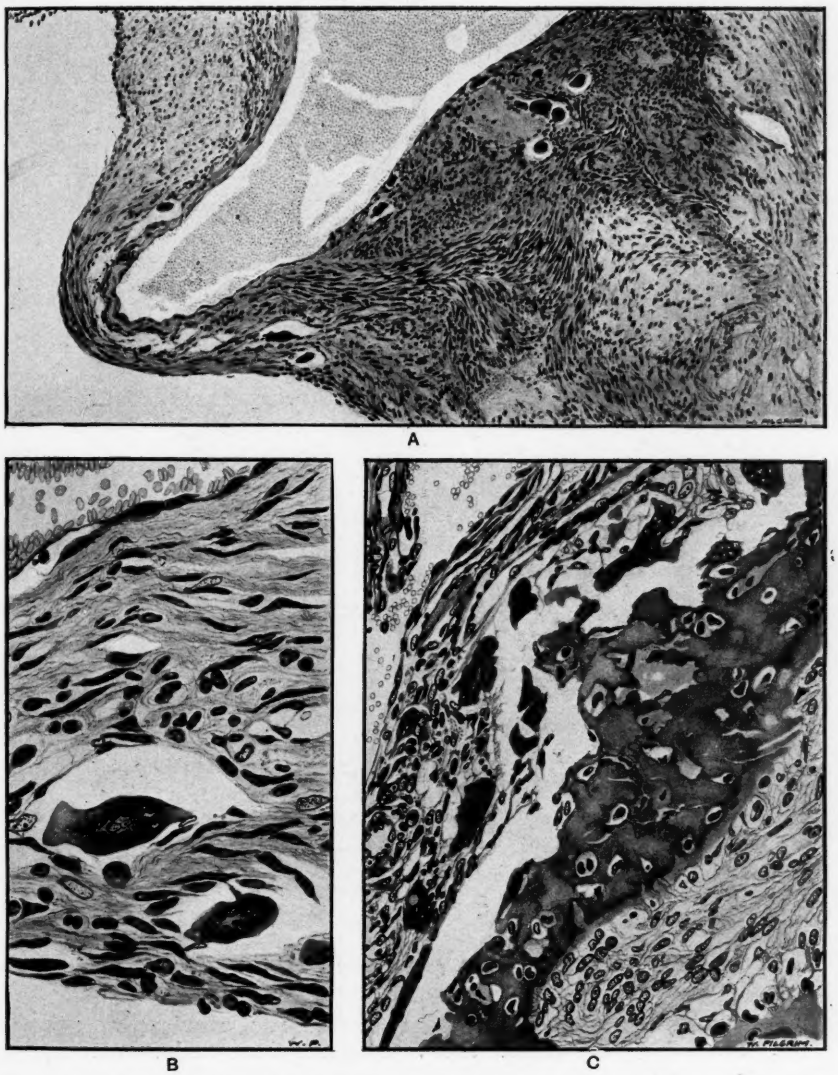

FIG. 457.—*Case* 2. Microscopic appearance of tumour shown in *Figs.* 455, 456. A, Low-power drawing of wall of blood-filled cyst in the tumour. B, Higher-power of portion of A, showing cyst wall with osteoclasts; C, Portion of tumour showing erosion of bone by osteoclasts.

SUBSEQUENT PROGRESS.—The wound healed by first intention, and convalescence was uninterrupted. The bladder required catheterizing after the operation for eighteen hours and then resumed normal function. There was no trouble with the bowels. The patient was discharged on the forty-ninth day after operation. A special webbing support taking the weight from the right shoulder was designed in consultation with Messrs. Ernst, and this is worn day and night. Five and a half years after the operation there is no sign of stretching of the abdominal parietes. As a young and active boy he had no difficulty in adapting himself

to new conditions, and was soon able to sit comfortably and to get about with the aid of crutches. He has refused offers of any endeavour to fit a prosthesis. He is now able to take an active part in the work of a busy butcher's shop in his native town; he drives a car, and is at present courting a sweetheart! (*Figs.* 455–458.)

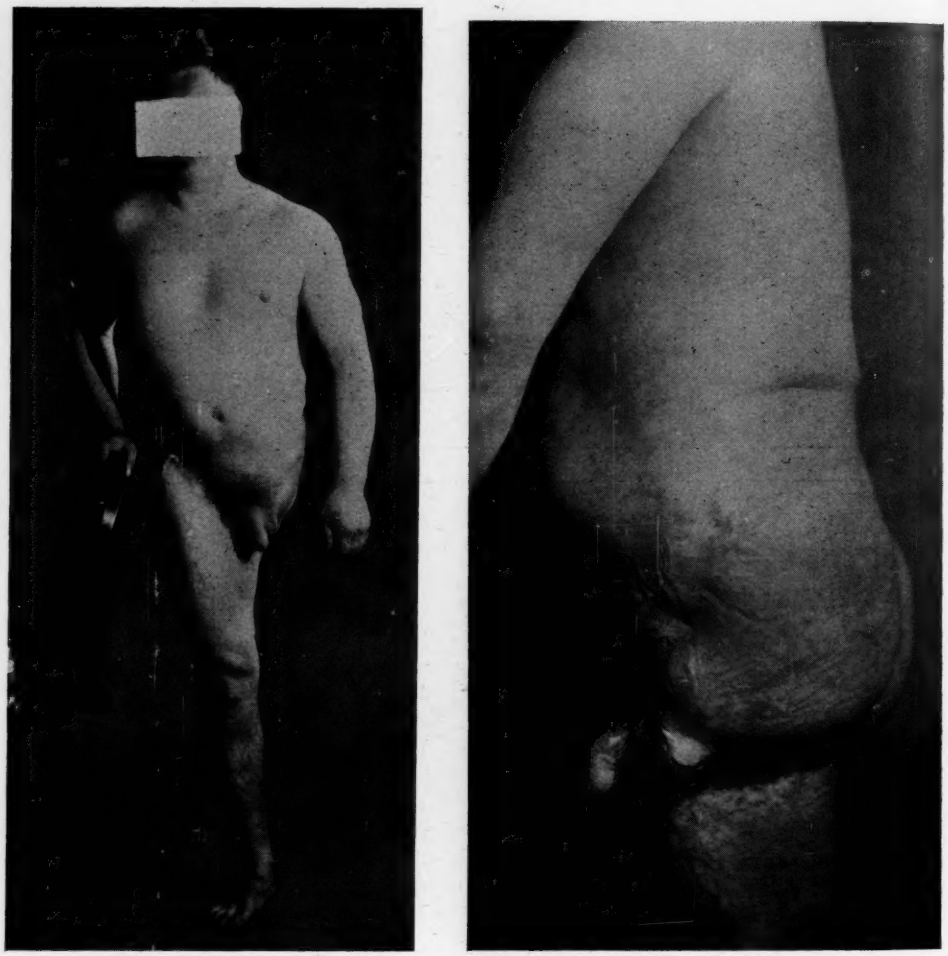

FIG. 458.—*Case* 2. Photographs of the patient taken five and a half years after operation.

Case 3.—**Enormous chondroma of innominate bone, overlapping upper end of femur and extending to costal arch. Interinnomino-abdominal amputation. Recovery.**

HISTORY.—H. S., male, aged 59, golf-course labourer. The patient had known of the presence of a "lump in the left groin" since infancy. This had gradually increased in size throughout his life, but during the two years preceding his admission the growth of the tumour had been rapid, and it had attained the enormous dimensions shown in the accompanying photographs (*Fig.* 459). The huge size of the mass had interfered with his walking for the past six months, and nine weeks previously he was compelled to give up his work on the golf-course. He could walk less than a hundred yards and had to stop by reason of the pain which he experienced. The pain was of a rheumatic character and radiated down the lateral aspect of the left thigh to the foot; although not always present, this pain was sometimes most severe, and before admission to the Middlesex Hospital its intensity had

necessitated the daily and nightly employment of veramon, nepenthe, etc., in increasing doses for its relief. He was virtually 'house-tied' by reason of his encumbrance and his sufferings.

The patient had been operated upon for a carcinoma of the lower lip in 1931; the growth had been excised and there was *no* evidence of any recurrence.

The enormous tumour of the left innominate bone, for which he claimed admission to hospital, reached to the left costal margin, pushing upwards and outwards the costal outlet; it extended downwards to the great trochanter, anteriorly almost to the umbilicus, and posteriorly it filled up the renal parallelogram. The mass was coarsely nodular, tightly

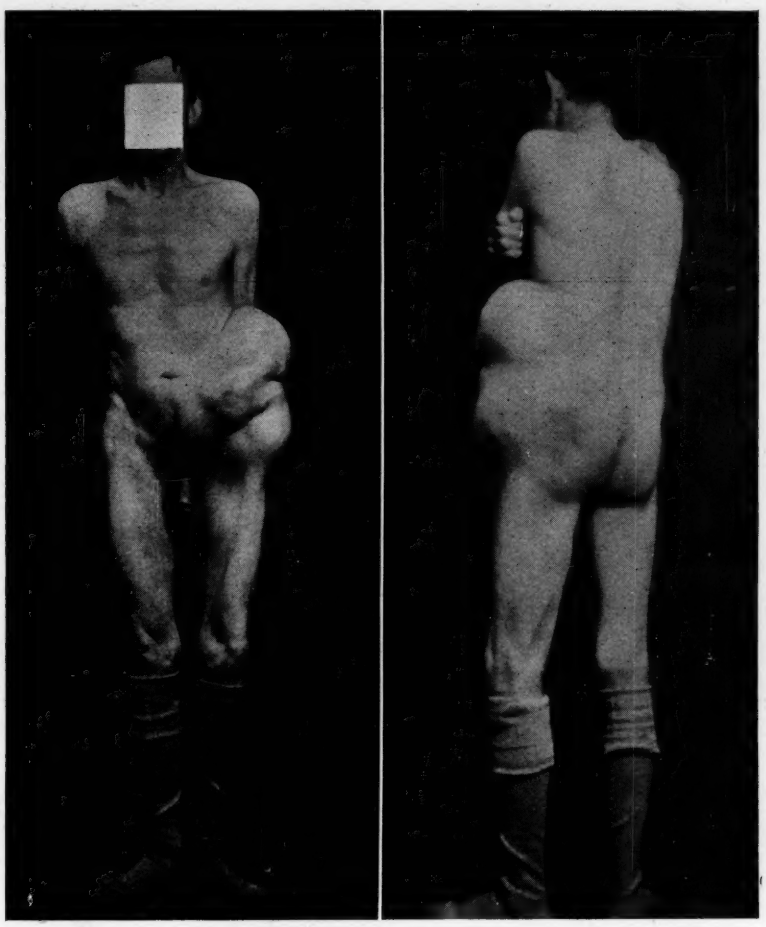

FIG. 459.—*Case* 3. Showing the extent of the colossal chondroma of the innominate bone and its upward and downward extension: anterior and posterior aspects.

stretching the skin; but the latter was not attached to it. The consistence of the tumour varied; in some parts it was cystic, in others very hard. There was well-marked calcification of the blood-vessels of the limbs, as demonstrated by radiography.

The anatomical connections precluded thorough removal of the tumour by any operative measure less severe than a hindquarter amputation; indeed, the manner in which the mass overlapped the great trochanter is well shown in *Fig.* 462.

The rapid growth of the tumour in the recent months which preceded his admission, and the pain experienced, cogently suggested a malignant transformation of an old-standing chondroma; moreover, it was felt that in the event of the tumour not being already malignant in nature, a more local, but imperfect, operation might expose the patient to the supervention

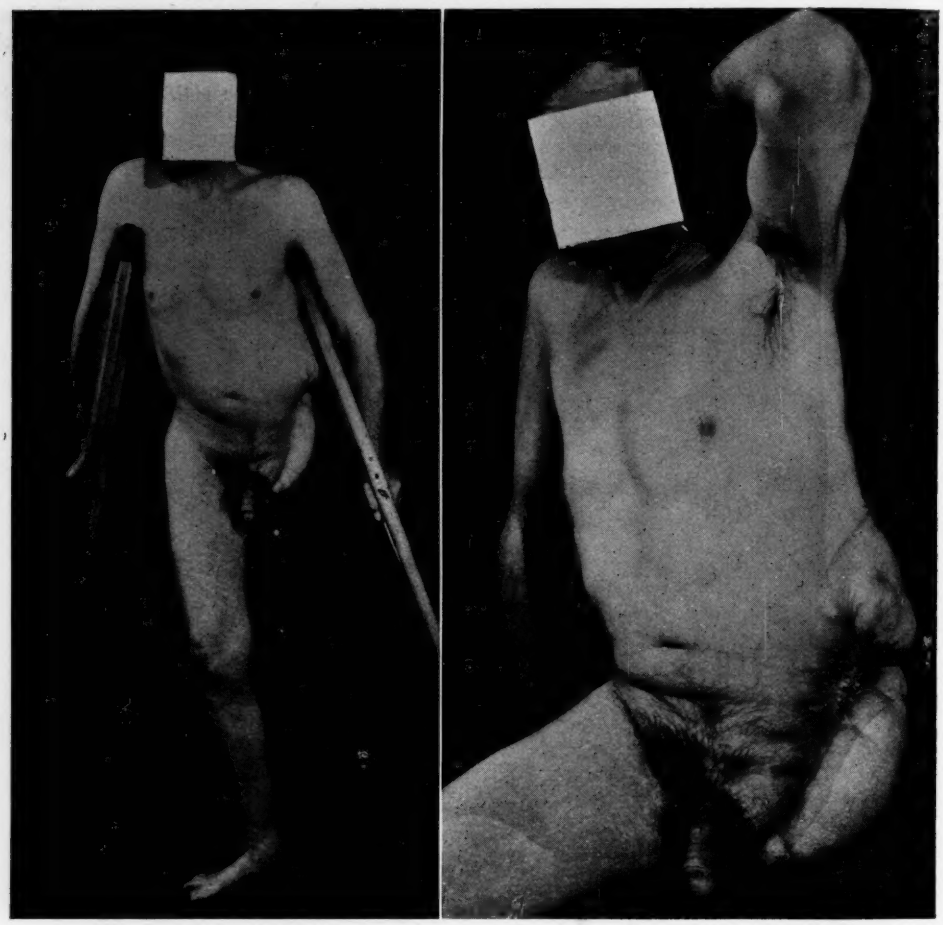

FIG. 460.—*Case* 3. Photograph of the patient nearly a year after operation.

FIG. 461.—*Case* 3. Photograph of specimen immediately after removal by hindquarter amputation.

of the sarcoma on the relics of a partially ablated chondroma—a calamitous experience which has indeed befallen one of us (G. G.-T.). It was therefore decided that an interinnomino-abdominal amputation held out the best hopes of a permanently successful issue, and, despite the calcified condition of his arteries, arrangements were instituted for its performance.

OPERATION (Nov. 5, 1933, G. G.-T.).—Messrs. Wiles, Banham, and Gowar gave most

FIG. 462.—*Case* 3. Chondroma of innominate bone. The muscles, etc., have been removed and the overlapping of the hip-joint and the upper end of the femur by the tumour is well shown. Anterior aspect.

valuable and loyal assistance, and Mr. E. G. Muir made himself responsible for such methods of intravenous therapy as appeared to be indicated during the operation. In point of fact, no less than 1200 c.c. of blood were transfused during the operation, and in addition 200 c.c. of saline were infused. Anæsthesia was partly by means of intrathecal injection of 2 c.c. of 5 per cent stovaine, and partly by means of gas and ether induction with a Clover

inhaler, followed by open ether. Mr. Idris most skilfully anæsthetized and courageously refused to exhibit any worry or alarm, even when in the middle of the operation the condition of the patient was desperate in the extreme.

The position and magnitude of the tumour had occasioned us some anxiety as to the possibility of having sufficient skin-flaps to cover in the large raw area; consequently an

FIG. 463.—*Case* 3. Same specimen as shown in *Fig.* 462, posterior aspect. The portion of sacrum removed with the innominate bone is shown.

internal flap from the adductor region of the thigh was employed according to the method of Savariaud (*see Fig.* 471,E) and the superficial femoral vessels were ligated low down so as to endeavour to secure the vitality of this flap. Apart from this region of the dissection, where the anatomy was undisturbed, the rest of the amputation was performed blindly, since the colossal tumour had obscured all landmarks, and there was no possibility of demonstrating the branches coming off the internal iliac artery and controlling these before they were cut.

The patient had to be moved on to his side and back again into the supine position on more than one occasion, with the result that an alarming fall of blood-pressure ensued which almost proved fatal. Fortunately there was ample blood in readiness for any instant transfusion, and its expeditious and timely employment undoubtedly staved off a disaster that was

FIG. 464.—*Case* 3. Section of specimen shown in *Figs.* 462, 463.

imminent. Later in the evening of Nov. 5 the patient was given another 500 c.c. of blood and an additional pint of saline infusion.

SUBSEQUENT PROGRESS.—The post-operative convalescence was by no means so uneventful as in *Case* 2, for the internal flap sloughed, and the wound everywhere healed badly. A

post-operative thrombo-phlebitis of the popliteal vein of the other limb also ensued, but despite all these anxieties the patient was able to go to a Convalescent Home on Jan. 12, 1934. (*Fig.* 460.)

The weight of the dried and macerated tumour, innominate bone, and upper end of femur was 12 lb. 4 oz. (*Figs.* 461–463). The section shows masses of hyaline cartilage with areas of ossification and cancellous bone (*Fig.* 464).

Case 4.—**Sarcoma of pelvis. Interinnomino-abdominal amputation. Death.**

HISTORY.—E. L., male, aged 28, had fallen from a lorry eighteen months before his admission into Middlesex Hospital. The accident occasioned considerable pain in the vicinity

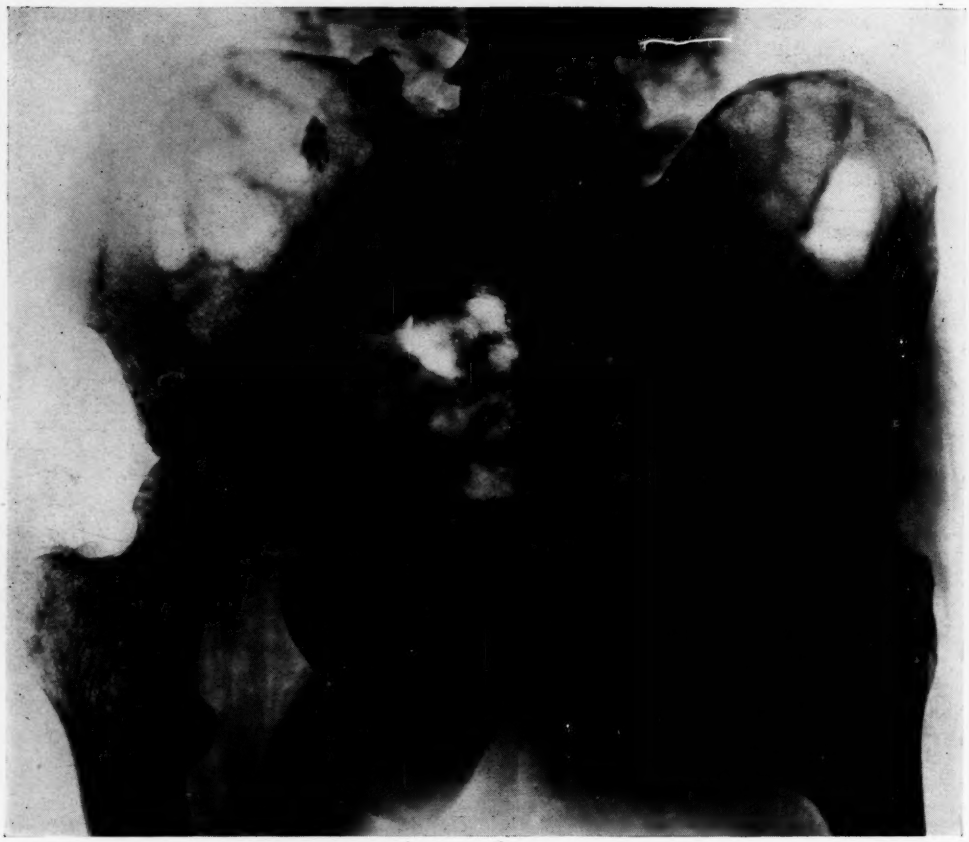

FIG. 463.—*Case* 4. Radiogram exhibiting the appearances of sarcoma of the pelvis.

of the left hip, which gradually passed off. A year ago the pain returned, and although the patient was at first regarded as a case of rheumatism, he was nevertheless X-rayed at a local hospital, where a diagnosis was made of tuberculosis of the left hip-joint. He was transferred to a sanatorium and treated by means of rest, splinting, and extension, but the appearance of a swelling in Scarpa's triangle led to the taking of another radiogram, which showed destruction of the ischium and pubes without any new bone formation. He was then seen in consultation by Mr. Ronald Reid, of Colchester, who at once diagnosed a malignant neoplasm, and he was straightway transferred to my care (G. G.-T.) in Middlesex Hospital.

ON EXAMINATION.—There was a hard, smooth mass present in Scarpa's triangle. It extended down the inner aspect of the thigh for about 4 in. and could be felt as an indefinite induration above the mesial end of Poupart's ligament. The tumour could also be felt in the ischio-rectal fossa, internal to the ischial tuberosity; there was no alteration in the position

of the anus. The left lower limb was maintained in a position of external rotation; the knee was slightly flexed. The femoral artery could be felt just lateral to the tumour. The case was regarded as a sarcoma, and in the absence of any evidence of metastasis, arrangements were made for amputation.

OPERATION (March 3, 1934, G. G.-T.).—Anæsthesia was induced by Mr. R. E. Apperly by means of intrathecal stovaine and a Clover induction, followed by open ether. Able assistance was rendered by Messrs. P. Wiles, Roy Banham, and Alan Kekwick. Mr. E. G. Muir kindly made himself responsible for the transfusions, as he had done in the preceding operation (*Case* 3).

The difficulties of this particular case were due to the fact that the growth had extended

FIG. 466.—*Case* 4. Specimen viewed from inner aspect. The inward projection of the mass is depicted: the latter proved to be somewhat adherent to the rectum and to the prostatic sheath.

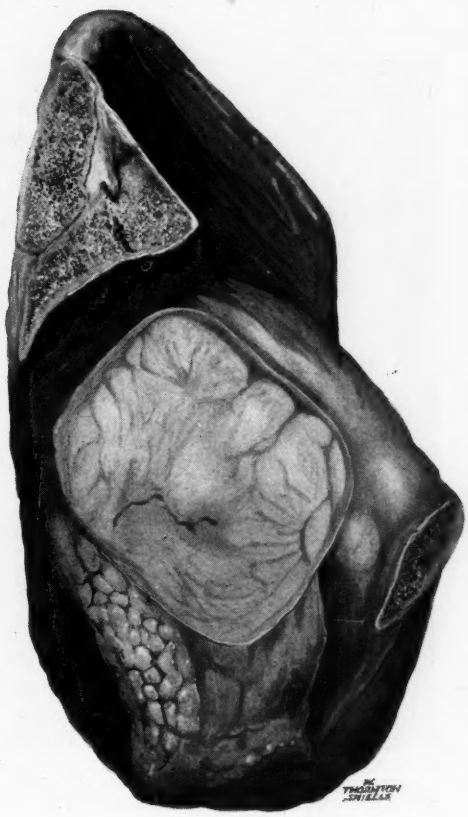

FIG. 467.—*Case* 4. Same specimen as shown in *Fig.* 466 on section, exhibiting naked-eye appearance of the neoplasm.

beyond the middle line, and the bone section had to traverse the innominate bone of the opposite side. The neoplasm was also somewhat adherent to the sheath of the prostate and the rectum, and had to be dissected off these structures. Apart from this there seemed nothing very remarkable to note; the operation was completed in sixty-five minutes and the patient left the theatre in good condition. A transfusion of 1200 c.c. of blood from two separate donors was performed during the operation. (*Figs.* 465–467.)

After his return to bed the patient began to go downhill and a further transfusion was given. This was unavailing, and death ensued two-and-a-half hours after the completion of the operation.

AUTOPSY.—At the autopsy there were no secondary growths anywhere throughout the body. The general musculature of the body was poor and anæmic and the cardiac muscle was especially soft and friable—possibly a consequence of the prolonged sojourn in bed for the treatment of his supposed tuberculosis. Some obsolete tuberculous mesenteric glands were found.

MICROSCOPIC REPORT.—The section showed a growth interspersed by strands of fibrous tissue and composed of large hyperchromated cells with scanty cytoplasm. There was very little intercellular substance, *no* alveolar arrangement, and no new bone formation. The appearance was suggestive of a Ewing's endothelial myeloma of bone.

Case 5.—**Sarcoma of the upper end of the femur. Interinnomino-abdominal amputation. Recovery.**

W. B., male, aged 18. Admitted for tumour of the upper thigh, which was alleged to been present for only three weeks.

FIG. 468.—*Case* 5. Photograph showing the relation of the tumour to the hip, negativing any amputation at that level.

ON EXAMINATION.—There was a large fusiform swelling at the junction of the upper and middle thirds of the right thigh; the tumour had almost attained the size of a Rugby football; it was firm but not tender. There were considerable veins coursing under the skin overlying the mass. There were no limitation of the movements of the lower limb. (*Fig.* 468.)

Radiological Report.—At the junction of the upper and middle thirds of the femur there was some rarefaction of the cortex, with periosteal reaction on both sides of the bone. On the posterior surface small spindles could be seen at right angles to the shaft. The appearances were those of a periosteal sarcoma.

The extent of the tumour, its position, and the involvement of the soft parts (*Fig.* 469) excluded any mere disarticulation at the hip-joint as an effective procedure if operation were decided upon; nothing less drastic than an interinnomino-abdominal amputation was likely to cure, if surgery rather than radiation therapy were contemplated. There was no evidence of any secondary growth in any other part of his body.

OPERATION (July 2, 1934, G. G.-T.).—Anæsthesia was by means of intrathecal stovaine (7 cgrm.) and also open ether; both were skilfully administered by Mr. Raymond Apperly. Messrs. P. Wiles, Alan Kekwick, and R. S. Handley rendered able assistance, and Mr. Lloyd-Davies kindly made himself responsible for any intravenous therapy that might be required during the operation.

Operation was simple, inasmuch as the innominate bone was nowhere invaded by new growth; the symphysis was cut through with a knife; the patient was, with the greatest gentleness, turned over toward the sound side, and to a degree that just sufficed to enable the dorsum ilii to be sawn across to the sciatic notch. It was then very easy to secure all the branches of the internal iliac vessels before they were cut, and the nerves were blocked with novocain before being divided. Every step in the operation was taken quickly and quietly; very little shock was produced, but it was deemed prudent at the very end to administer a blood transfusion of 500 c.c. The operation lasted an hour and ten minutes, and the condition of the patient at the end was surprisingly good, reminding all of *Case* 2 in this series in the excellence of his state at the termination of so severe an amputation. The pulse-rate on the return of the patient to bed was 84. Recovery was uneventful, and the wound healed by first intention.

PATHOLOGICAL REPORT.—On section a spindle-shaped tumour arising from the periosteum of the upper half of the femur, chiefly from the inner aspect (transverse diameter, 5 in.). The growth is soft, with areas of hæmorrhage, and is invading the overlying muscles; there is no evidence of bone formation, and little evidence of invasion of the bone cortex. *Spindle-celled sarcoma.* (*Fig.* 470.)

Section shows infiltration of tissues by solid masses of darkly staining short-spindle cells. There is no evidence of bone formation.

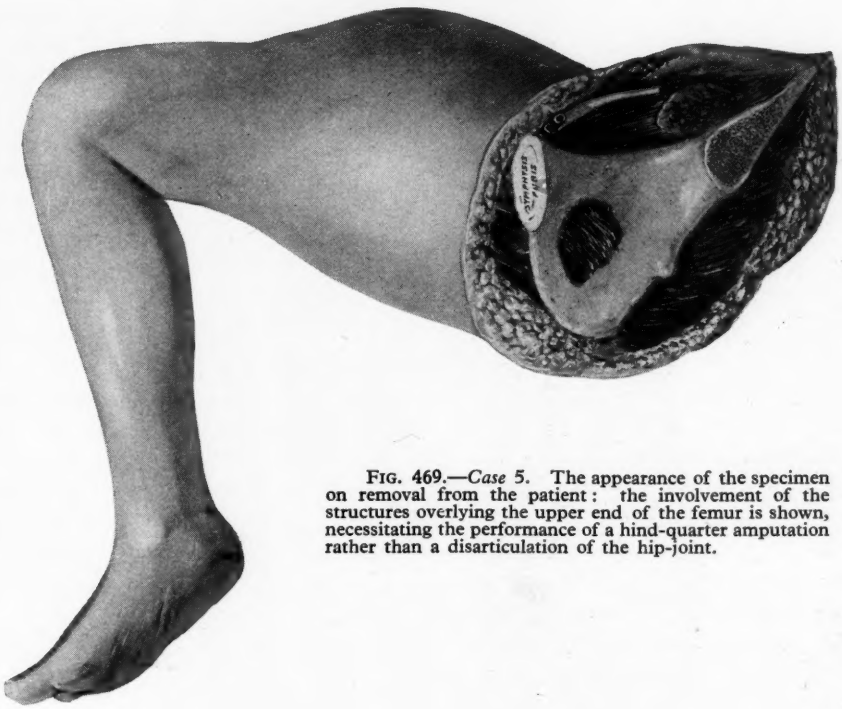

FIG. 469.—*Case* 5. The appearance of the specimen on removal from the patient: the involvement of the structures overlying the upper end of the femur is shown, necessitating the performance of a hind-quarter amputation rather than a disarticulation of the hip-joint.

TECHNIQUE OF THE OPERATION

It is significant of the excellence of Hogarth Pringle's description of this amputation that his account[3] has been re-read by one of us (G. G.-T.) before the performance of each of the operations on the cases narrated in the preceding pages.

Anæsthesia.—In each case spinal anæsthesia was employed in conjunction with general anæsthesia; the latter was induced by means of gas and ether in a Clover inhaler, and this was followed by open ether. *Dosage of stovaine*: probably about 7 cgrm. stovaine is an appropriate dosage, but in *Case* 3 an injection of 10 cgrm. was administered.

Incision.—Several incisions have been employed by various surgeons (*Fig.* 471). In four of the cases detailed above the incision was that recommended by Hogarth Pringle: this is a modification of Girard's incision and will be found to satisfy the requirements of most cases, save when the anatomy of the pelvis is grossly deformed by gigantic tumours, such as that depicted in *Fig.* 459 (*Case* 3). In this case the incision of Savariaud was made use of, and a long

internal flap employed. Though every endeavour was made to ensure a good blood-supply to the flap, it sloughed in part. Possibly the degenerated state of the arterial system may have accounted for this.

FIG. 470.—*Case* 5. Specimen of sarcoma of the upper end of the femur on section: showing relation to the overlying muscles.

Position.—The patient is turned slightly on to the sound side, with a sand-bag under the shoulder and another under the lower part of the thigh of the affected side. While these dispositions are being made *one of the veins* of the antecubital fossa of

the upper extremity of the sound side is *rapidly exposed*, with a view to facilitate the transfusion of blood, for which previous arrangements have been organized.

The incision is carried along the crest of the ilium from behind the posterior superior spine to the anterior superior spine of that bone: it is then continued downwards and inwards 1½ in. below Poupart's ligament towards the middle of the origin of the adductor brevis. Where any doubt is experienced as to the fixity of the neoplasm on the inner aspect of the innominate bone, and as to the possibility of removal, it is well straightway to cut through the flat muscles of the abdominal wall at or above their attachments to the iliac crest, and to satisfy oneself at once as to the pathological integrity of the peritoneum, etc., before proceeding to deal with the external iliac vessels. Where no such doubts are felt, it may be of some comfort to the operator to deal with the external iliac vessels as soon as this first incision is made. Except in *Case* 3, where the femoral vessels were ligated, the external iliacs have been secured at the level of Poupart's ligament. *No* direct search was made for deep epigastric or deep circumflex iliac vessels; and they do *not* appear to have been damaged.

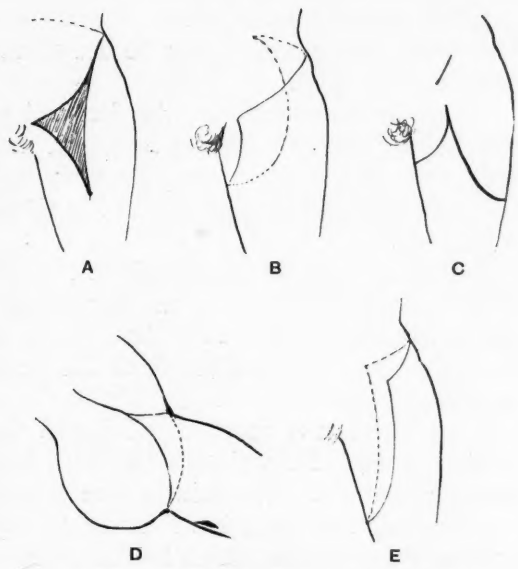

FIG. 471.—Diagram showing some of the various incisions that have been employed in the operation of interinnomino-abdominal amputation. A, Jaboulay; B, Girard; C, Bardenheuer; D, Salistcheff; E, Savariaud.

FIG. 472.—Diagrammatic sketch indicating one stage in the operation: Poupart's ligament has been divided at each end, and the inner pillar of the external abdominal ring has been cut through, enabling the operator to retract the spermatic cord inwards and to get access to the insertion of the rectus abdominis. The rectus and the symphysis are now divided.

Poupart's ligament is divided at each end (*Fig.* 472), and the inner pillar of the external abdominal ring, thus allowing the spermatic cord to be drawn inward out of harm's way. The insertion of the rectus abdominis to the pubic crest is next divided, and, after the anterior and posterior surfaces of the pubic bone have been cleared by blunt dissection, the symphysis pubis is divided by knife, or if necessary by saw (*Fig.* 473).

The patient is then very gently turned further over on to the sound side, and an incision carried from the mid-point of the iliac crest down to the gluteal fold, which is followed to meet the lower and mesial extremity of the first incision in the cruro-scrotal sulcus. The dorsum ilii is exposed and sawn through into the sciatic

notch: this manœuvre is more simple and expeditious than attempting to disarticulate at the sacro-iliac synchondrosis. If it be necessary, the small piece of the auricular surface of the ilium can be rapidly detached afterwards.

The anterior and posterior bone sections being now complete, the innominate bone and lower extremity can be drawn away from the pelvic peritoneum and its contents (*Fig.* 474).

The lumbo-sacral cord, the first and second sacral nerves, and the obturator can all be identified and injected with novocain before division. The obturator artery, the gluteal artery, the sciatic artery, and probably the internal pudic artery, can be secured before division, and in this manner the hæmorrhage can be greatly minimized.

The psoas is cut through above the pelvic brim, and the division of the pyriformis and the levator ani, and the detachment of the ischiocavernosus and crus penis from the ischiopubic ramus liberates the innominate bone from the last fibromuscular links which fetter it to the underlying structures; the limb and the innominate bone are free.

All bleeding is now controlled, and the peritoneum reinforced by the suture of the remains of the gluteus maximus, levator ani, etc., to the flank muscles and rectus abdominis. The skin is then sutured with interrupted stitches.

Retention of urine has followed the operation in the three of our cases that survived, and catheterization has been required.

The most important points in the conduct of the operation would appear to be: (1) The most gentle care should be taken in turning the patient: the danger from rough or excessive movement of a patient under spinal anæsthesia cannot be exaggerated. (2) The most thorough organization of blood transfusion arrangements. (3) Division of the posterior portion of the dorsum ilii with the saw is more expeditious and simple than disarticulation of the bone at the sacro-iliac synchondrosis; it is easy subsequently to ablate the auricular portion of the ilium. (4) Hogarth Pringle's incision is that most universally satisfactory; (5) It is almost unnecessary to stress the importance of minimizing hæmorrhage and shock by preliminary control of vessels before division, and of nerve-blocking before section of the trunks.

STATISTICS

One of us (P. W.) has expended much time in bringing the literature of the subject up to date, and, including the 5 personal cases placed on record in this communication, has verified the references in 55 cases in which the innominate

Table I.—Operative Mortality in Interinnomino-abdominal Amputation

Nature of Disease	Number of Cases	Recovery	Death	Percentage Mortality
Sarcoma	37	17	20	54·1
Tuberculosis	15	5	10	66·6
Non-tuberculous disease of innominate bone and hip-joint	1	1	—	—
Osteoclastoma	1	1	—	—
Unknown origin	1	—	1	—
Total ..	55	24	31	56·4

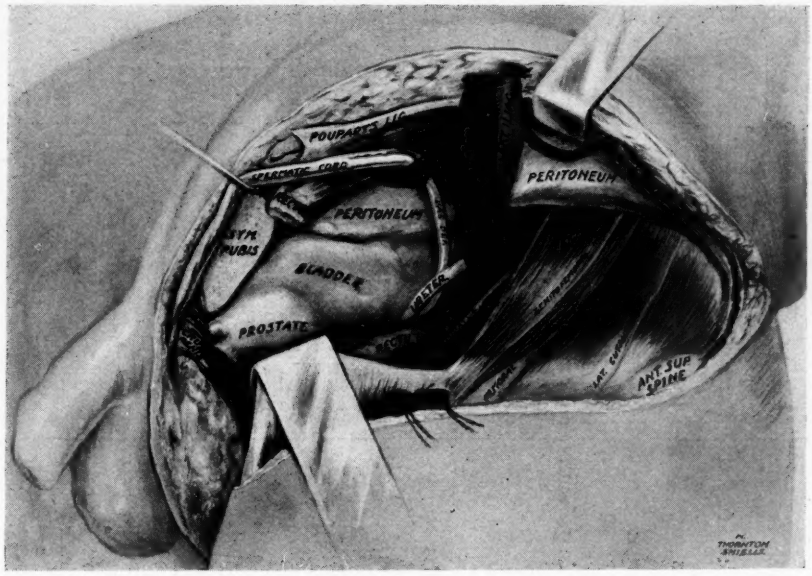

FIG. 473.—Anatomical structures displayed during interinnomino-abdominal amputation. The external iliac vessels have been ligatured distal to the origin of the epigastrics; the symphysis has been divided, and the innominate bone is being retracted outwards.

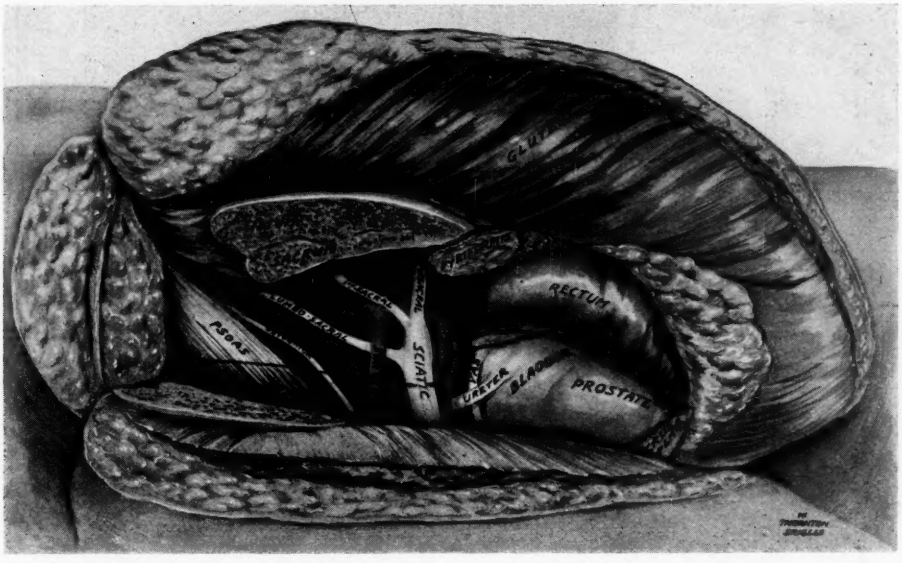

FIG. 474.—The patient has been turned over on to the sound side and the dorsum ilii has been sawn through: the symphysis pubis having been divided, the innominate bone is being drawn away from the pelvic peritoneum and the pelvic viscera. The arteries and nerves which require surgical attention or division are shown. The line of section of the psoas is indicated.

Table II.—Details of Interinnomino-Abdominal Amputations Recorded in the Literature (55 Cases)

Surgeon and Date	Sex and Age	Disease	Result	Remarks
Abrashanaw, A. (1930) ..	F. 35	Sarcoma	Lived at least 3 months	Reported by S. I. Riswach (*Deut. Zeits. f. Chir.*, 1932–3, ccxxxviii, 121)
Axhausen, O. (1910) ..	F. 45	Sarcoma	Died in 9 hours	Momburg's tourniquet. Cut ilium and pubis and joined with graft (*Arch. f. klin. Chir.*, 1910, xci, 538)
Bardenheuer (1897) ..	F. 46	Tuberculosis	Alive 10 months later	*Zentralb. f. Chir.*, 1897, xxiv, supl., 132; also *Verh. Deut. Gesel. f. Chir.*, 1897, xxvi, 130. Reported by O. Wolff, *Zentralb. f. Chir.*, 1897, xxiv, 185
Babcock, W. W. (1916) ..	M. 52	Sarcoma	Died of shock soon after operation	Three previous local operations (*Surg. Gynecol. and Obst.*, 1918, xxvi, 554)
Bier (1909)	? ?	Sarcoma	Lived 2 months	Momburg's tourniquet (*Deut. med. Woch.*, 1909, xxxv, 1, 45)
Billroth (1891)	? ?	?	Died in few hours	Savariaud says that Berg, of Stockholm, told Girard he was assistant to Billroth when he performed this operation
Cacciopoli, G. (1894) ..	M. 17	Sarcoma	Died of shock in 3 hours	*Riforma Med.*, 1894, ii, 819 and 831
Christel (1903)	F. 2	Tuberculosis	Lived	Reported by Dreist (*Deut. Zeits. f. Chir.*, 1904, lxxi, 4)
Faure, J. L. (1899) ..	F. 16	Sarcoma	Died next day	Temporary ligature aorta. Operation abandoned because of hæmorrhage (*Rev. de Gynecol. et de Chir. abdom.*, 1899, iii, 713)
Freeman, L. (1899) ..	F. 38	Sarcoma	Lived 20 months	First disarticulated hip, then removed a lot of pelvis (*Ann. of Surg.*, 1901, xxxiii, 318)
Gallet (1900)	M. 25	Tuberculosis	Died in 6 hours	Disarticulation of hip 2 years before (*Jour. de Chir. (Belge)*, 1901, i, 569)
Gallet (1901)	F. 39	'Telangiectatic' sarcoma	Died in 1 hour	Disarticulation of hip 2 years before (*Ibid.*)
Gaudier, H. (1913) ..	M. 15	Non-tuberculous osteomyelitis	Lived	Momburg's tourniquet. Disarticulated hip-joint (*Bull. et Mém. Soc. Chir. de Paris*, 1913, xxxix, 1570)
Girard (1895)	F. 17	Sarcoma	Lived; 7 months later had secondary in scar	Disarticulation of hip the year before (*Congrès franç. de Chir.*, 1895, ix, 823)
Girard (1895)	M. 15	Tuberculosis	Died in 5 minutes	Previously had resection of knee (*Ibid.*, 1898, xii, 585)
Girard (1897)	M. 52	Sarcoma	Lived 6 months and died of secondaries	*Ibid.*
Gordon-Taylor, G. (1922) ..	M. 25	Sarcoma	Died of shock in 6 hours	
Gordon-Taylor, G. (1929) ..	M. 17	Osteoclastoma	Well 5½ years later	
Gordon-Taylor, G. (1933) ..	M. 59	Chondrosarcoma	Lived	
Gordon-Taylor, G. (1934) ..	M. 28	Sarcoma	Died in 2½ hours	
Gordon-Taylor, G. (1934) ..	M. 18	Sarcoma	Lived	
Judin, S. S. (1924) ..	F. 36	Chondrosarcoma	Lived at least 2½ years	Some months after operation had a child with easy labour (*Surg. Gynecol. and Obst.*, 1926, xliii, 668)
Jaboulay (1894)	? ?	Sarcoma	Died	Patient *in extremis* at time of operation (*Lyon méd.*, 1894, lxxv, 507)
Jaboulay (1895)	M. 66	Sarcoma	Died 36 hours from infection	Reported by G. Gayet (*Province méd.*, 1895, xxxiv, 397)
Kadjan, A. (1901)	M. 25	Sarcoma	Died of shock in 2 days	Poor condition: metastases found post mortem (*Ljstopis Russ. Chir.*, 1900, iv; and in *Jahrb. v. d. Forts. d. Chir.*, 1901, vii, 1103)
Keen, W. W., and da Costa,	M. 42	Sarcoma	Died in 33 hours	Sawed through pubis and ilium (*Internat.*

Keen, W. W., and da Costa, J. C. (1903)	M. 42	Sarcoma	Died in 33 hours	Sawed through pubis and ilium (*Internat. Clin.*, iv, s. 13, 127)
Krynski (1908)	? ?	Sarcoma	Lived 5 weeks; died of cachexia	*Przeglad. Chir. i. Gynck.*, 1909, i, 13, abstr. in *Zentralb. f. Chir.*, 1910, xxxvii, 1132
Lastaria, F. (1906)	M. 22	Chondrosarcoma	Died during operation	*Riforma Med.*, 1907, xxiii, 457
Loeffler, F. (1918)	M. 11	Sarcoma	Lived at least 15 months	*Zeits. f. orthop. Chir.*, 1919, xxxix, 305
Maffei, A. (1915)	F. 5	Tuberculous hip	Lived. Well 17 years after	Numerous fistulæ, high temperature. Momburg's tourniquet. X-rays and photographs taken in 1932 (*Proc. Soc. Internat. de Chir. orthop.*, 1933, 270)
Maffei, A. (1915)	M. 8	Tuberculous hip, cachectic, fistulæ	Lived one month	Recovered from immediate effects of operation. Temperature did not fall; disease continued its course
Maffei, A. (1915)	? ?	(Child) tuberculous hip	Died in 2 hours	"Le Traitement de la Coxite tuberculeuse", by A. Maffei (Bruxelles): *2me Congrès internat. de Chir. orthop.*, Londres, 1933, p. 270
Morestin (1902)	F. ?	Sarcoma	Died in 9 hours	Operation abandoned before finished (*Bull. Soc. Anat. de Paris*, 1902, iv, s. 6, 795)
Morestin (1905)	M. ?	Tuberculosis	Lived at least 3 years	Amputation of hip 1 year before. Very emaciated. Open sinuses (*Bull. Soc. Chir. Paris*, 1908, xxxiv, 1060)
Morestin (1909)	M. 35	Tuberculosis	Died in 5 hours	Leg amputated at hip 2 months earlier. In this and 1905 case the tuber ischii was left (*Bull. Soc. Anat. de Paris*, 1909, xi, s. 6, 288
Nanu (1900)	M. 50	Myxosarcoma	Died in 20 days of gangrene flaps—tied common iliac vessels	*Congrès internat. de Méd.*, Paris, 1900 (Sec. Chir. gén.), 511
Orlow, L. (1907)	M. 47	Sarcoma	Died 35th day of sepsis of wound	*Westnik. Chir.*, 1907, ii, 225, abstr. in *Jahrb. v. d. Forts. d. Chir.*, 1907, vii, 1104
Pagenstecher, E. (1909) ..	F. 50	Sarcoma	Lived at least 3 months	Momburg's tourniquet (*Arch. f. klin. Chir.*, 1909, xc, 160)
Roux (1909)	M. 40	Sarcoma	Lived at least 3 months	Reported by G. Plantard (*Arch. Prov. de Chir.*, 1909, xviii, 657)
Pringle, H. (1900)	F. 10	Tuberculosis	Lived 14 years	Amputation of hip and removal of much of pelvis (*Lancet*, 1909, i, 530)
Pringle, H. (1906)	M. Adlt.	Tuberculosis	Died in 14 hours	Multiple sinuses (*Ibid.*)
Pringle, H. (1908)	M. 18	Sarcoma	Lived 5 months, died of secondaries	Amputation of leg 2 years before (*Ibid.*)
Pringle, H. (1915)	M. 34	Sarcoma	Lived	*Brit. Jour. Surg.*, 1916, iv, 283
Pringle, H. (1915)	F. ?	Sarcoma	Lived	*Ibid.*
Ransohoff, J. (1909) ..	M. 45	Sarcoma	Died in 4 weeks from 'entero-colitis'	Poor condition, multiple sinuses (*Ann. of Surg.*, 1909, l, 925)
Ribera, Y. S. (1902) ..	? 8	Tuberculosis	Died in 8 days from gangrene of flaps. Tied common iliac artery	Reported by Luis y Simón, *Siglo méd.*, 1903, l, 779
Ribera, Y. S. (1902) ..	? 8	Tuberculosis	Died soon after operation	*Ibid.*
Ribera, Y. S. (1902) ..	F. 10	Tuberculosis	Died 3 hours after operation	*Ibid.*
de Ruyter (1902)	M. 45	Sarcoma	Died in 1 hour	H. Mayer, *Inaug. Dissert.*, Leipzig, 1902
Salistcheff (1898)	M. 38	Sarcoma	Lived. Tied common iliac artery, healing by second intention	*Arch. f. klin. Chir.*, 1900, lx, 57
Savariaud (1901)	F. 7	Sarcoma	Died same day of 'syncope'	*Rev. de Chir.*, 1902, xxvi, 345
Schadelmose, V. (1923) ..	? ?	Chondrosarcoma	Lived at least 10 months	*Acta. Scand. Chir.*, 1924, liii, 523. Momburg
Speed, Kellog (1932) ..	M. 59	Sarcoma	Died at end of operation	Secondaries lungs (*Ann. of Surg.*, 1932, xcv, 167)
Speed, Kellog (1932) ..	M. 45	Sarcoma	Died in 1 hour	*Ibid.*
Verneuil (1905)	? ?	Tuberculosis	Died in 2 hours	*Jour. de Chir. et Ann. Soc. belge de Chir.*, 1905, v, 406. Cited by Pringle, *Brit. Jour. Surg.*, 1916, iv, 283

Table III.—Chronological Table of Operations and Surgeons

1891.	Billroth	Died
1894.	Cacciopoli	Died
	Jaboulay	Died
1895.	Girard	*Lived*
	Girard	Died
	Jaboulay	Died
1897.	Bardenheuer	*Lived*
	Girard	*Lived*
1898.	Salistcheff	*Lived*
1899.	Faure	Died
	Freeman	*Lived*
1900.	Gallet	Died
	Nanu	Died
	Pringle	*Lived*
1901.	Gallet	Died
	Kadjan	Died
	Savariaud	Died
1902.	Morestin	Died
	Ribera	Died
	Ribera	Died
	Ribera	Died
	de Ruyter	Died
1903.	Christel	*Lived*
	Keen	Died
1905.	Morestin	*Lived*
	Verneuil	Died
1906.	Lastaria	Died
	Pringle	Died
1907.	Orlov	Died
1908.	Pringle	*Lived*
	Krynski	*Lived*
1909.	Bier	*Lived*
	Morestin	Died
	Pagenstecher	*Lived*
	Ransohoff	Died
	Roux	*Lived*
1910.	Auxhausen	Died
1913.	Gaudier..	*Lived*
1915.	Pringle	*Lived*
	Pringle	*Lived*
	Maffei	*Lived*
	Maffei	Died
	Maffei	Died
1916.	Babcock	Died
1918.	Loeffler	*Lived*
1922.	Gordon-Taylor	Died
1923.	Schadelmose	*Lived*
1924.	Judin	*Lived*
1929.	Gordon-Taylor	*Lived*
	Riswach	*Lived*
1930.	Abrashanaw	*Lived*
1932.	Speed	Died
	Speed	Died
1933.	Gordon-Taylor	*Lived*
1934.	Gordon-Taylor	Died
	Gordon-Taylor	*Lived*

Table IV.—Cases from the "Russian Literature"

Surgeon and Date	Sex and Age	Disease	Result	Remarks
Orlow (1901) ..	M. 47	Sarcoma	D	*Messenger of Surgery* (Russian), 1901, No. 2
Michailow (1902)	M. 17	Tuberculosis	D	*Surgery* (Russia), 1902, v, xi
Sinakevitch (1908)	M. 47	Sarcoma	D	*Surgery* (Russia), xxvii, p. 60
Veinshall (1921)	M. 25	Sarcoma	D	*New Surg. Arch.* (Russia), iii, 147
Bergman (1918)	? ?	5 non-T.B. osteomyelitis	2 D, 3 L	Quoted by Judin from Veinshall
Bryosovski (1921)	F. 16	Sarcoma	L	*New Surg. Arch.*, iv, 179
Czerny Goldberg ..				Cited by Bryosovski; no details given or results
Israel	F. ?	Sarcoma	D	Cited by Bryosovski
Smirnov (1919)	M. 33	Tuberculosis	D	Cited by Bryosovski
Toprover-Krimov	M. 47	Sarcoma	L 2 months	*Ekaterinoslav Med. Jour.*, 1924, i, 44
Rabinovitch ..	M. 58	Sarcoma	L 5 weeks	*New Surg.*, 1925, iii, 374
Rabinovitch ..	M. 49	Sarcoma	D	*New Surg.*, 1925, iii, 374
Krassintzev ..	M. 43	Sarcoma	L	*Moscow Surg. Soc.*, 1925, xii

D = Died. L = Lived.

Table V.—Personal Communications to Judin

Kokin ..	F. 30 ..	Sarcoma ..	Died
Rosanov ..	F. 27 ..	Tuberculosis	Died
Rosanov ..	M. 35 ..	Sarcoma ..	Lived 3 months
Rosanov ..	M. 30 ..	Sarcoma ..	Died
Krassintzev ..	M. ? ..	Sarcoma ..	Died
Krassintzev ..	M. ? ..	Sarcoma ..	Died
Krassintzev ..	M. ? ..	Sarcoma ..	Died
Krassintzev ..	M. 45 ..	Sarcoma ..	Died

bone and lower limb have been removed, either as a single-stage or as a plurigrade amputation. In some of the cases included in this series the removal of the innominate bone has been incomplete.

Of these 55 cases, 43 complete amputations have been performed as a primary operation. These cases are analysed in *Table I.*

It will be seen that the mortality of the procedure in the cases that we have been able definitely to verify, has been 56·4 per cent. *Tables II* and *III* give details of cases found in the literature.

A close scrutiny of the 74 cases reported by Judin[4] in 1926 reveals the fact that no fewer than 9 of these cases were duplicates, that another patient is reported to have died whereas the case ended successfully, and there are other inaccuracies which need not be elaborated here. A certain lack of confidence is naturally created, and it becomes difficult to assess the degree of credence that should be given to those reports from the "Russian literature", to which we ourselves have been unable to gain access. (*Tables IV, V.*)

If we add these "Russian figures" to those that we have confirmed and verified, there are some additional 24 cases with 16 deaths; 16 of these operations were for sarcoma, of whom 11 succumbed to the operation.

The operation has therefore been done about 79 times, with 46 deaths, i.e., a mortality-rate of 59·5 per cent.

In judging these figures it must be remembered that unsuccessful cases are much less likely to see the light of day than are successful ones; this statement is more particularly true at the present time, when the operation is an established procedure, than formerly, when even an attempted operation of this character was indeed an achievement of note. It is almost certain, therefore, that the mortality-rate given here underestimates the real gravity of the operation.

SUMMARY

1. A short note is given of five interinnomino-abdominal or hindquarter amputations, of which three were successful. Of the two fatal cases, one undoubtedly died from a failure in the transfusion arrangements, such as would not occur to-day; in the other fatal case, death appeared to have been due to cardiac failure. In this patient it was perhaps not sufficiently appreciated how baneful an effect upon the heart muscle and on the tissues of the body had been the very long confinement to bed on a mistaken diagnosis.

2. The operation will always remain one of the most colossal in surgery, but organization of team-work and the perfecting of transfusion arrangements will bring the mortality of the amputation down to a still lower percentage.

3. An attempt has been made to bring the literature of the operation up to date.

4. A description of the operation has been included.

For the beautiful pictures which adorn this paper we have to thank our friend Mr. Thornton Shiells.

REFERENCES

[1] BILLROTH, Quoted by Savariaud, *Rev. de Chir.*, 1902, xxvi, 345.
[2] GIRARD, *Congrès franç. de Chir.*, 1895, ix, 823.
[3] PRINGLE, H., *Brit. Jour. Surg.*, 1916, iv, 283.
[4] JUDIN, *Surg. Gynecol. and Obst.*, 1926, xliii, 668.

THE PATHOLOGY OF CONGENITAL GENU RECURVATUM*

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THE striking deformity which has been called in the past 'congenital dislocation of the knee' or 'luxation congénitale du tibia en avant', but which is now more accurately described as 'congenital genu recurvatum', has been known to surgeons and orthopædists since 1822, when, according to Potel, Chatelain, a Swiss observer, placed a case on record. I have been unable to verify the reference personally (*Bibliothèque médicale*, 1822, lxxv, 103). Since that time many cases have been described, and in 1897 Potel[64] gave a masterly description of the malformation in his thesis on deformities of the knee. At that date he was able to give abstracts of no fewer than 78 cases, which constituted the literature of the subject since the first case recorded by Chatelain. Since the publication of Potel's thesis nothing appears to have been added to our knowledge of the pathological processes involved in the production of the deformity, though I have been able to collect *personally* verified references to 80 fresh cases, giving a total of 158 cases.

Briefly, the malformation has in all recorded cases consisted of a fixed hyperextension of the leg at the knee-joint. The anterior aspect of the joint shows transverse creases in the skin, and the patella is small or may be absent. Posteriorly the hamstring muscles appear to be stretched and the femoral condyles may be felt projecting in the popliteal fossa. The deformity may be increased to some extent by manipulation, but on attempting to flex the joint a distinct elastic resistance is appreciated. In some of the cases on record manipulation succeeded in overcoming the resistance without much difficulty, but it should be noted that this occurred before the introduction of radiography, and it will appear from the case I am about to describe that the reposition which some of the older authors claimed may have been only apparent. In the majority of cases a slight degree of flexion can be produced by manipulation, but on realeasing the limb the leg at once springs back into the abnormal position. In the few cases where dissection has been carried out it has been noted that the articular surfaces are invariably normal in every respect. Tarnier,[86] indeed, observed that the stretched hamstring muscles might pass anterior to the femoral condyles and act as extensors at the knee-joint. It is certain, however, that this is the result of the deformity and not its cause.

The deformity may be unilateral or bilateral. It may be accompanied by isolated contractures elsewhere or by more or less generalized contractures of the limbs. It shows an especial tendency to be associated with club-foot and with congenital dislocation at the hip of the deformed limb.

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ETIOLOGY

Three main theories have been advanced to explain the malformation.

1. **The Theory of Uterine Compression.**—It is not perhaps unnatural that it should have been supposed that fixation of the knee-joint with the leg in hyperextension during intra-uterine life, and with the foot of the fœtus hooked over the shoulder, was responsible for the fixed hyperextension persisting into post-natal life. Uterine compression has for many years been blamed for almost every known congenital deformity, though there is clear evidence in the writings of the last generation that such a theory has failed to satisfy those who had seriously considered the question. I have had the opportunity elsewhere of stating my reasons for believing that uterine pressure is never the cause of real and persistent deformities.[54]

2. **The Theory of Epiphysial Separation.**—Phocas[62] believed that the deformity was not present actually at the joint, but that the hyperextension resulted from a forward displacement of the distal femoral epiphysis resulting from intra-uterine injury. His theory was accepted for a short time and good results were recorded from "manual osteoclasis of the femur". Dissections and the advent of radiography have proved his theory to be wrong.

3. **The Theory of Muscular Contracture.**—Potel[64] arrived at almost complete understanding of the problem when he pointed out that the hyperextension was entirely due to contracture of the quadriceps extensor muscle group gradually dragging the knee into a deformed position during intra-uterine growth. He pointed out that in all severe cases tenotomy of the quadriceps tendon was necessary before reduction could be obtained, and suggested that the 'manual osteoclasis' of the older authors was merely a subcutaneous rupture of the quadriceps or patellar tendons. Owen,[60] in 1891, had the distinction of being the first surgeon to carry out an open division of the quadriceps tendon in order to bring about reduction, and Potel in discussing this case makes the following remark: "If the old adage be true 'Naturam morborum ostendunt curationes', this single observation suffices to show that the cause of the hyperextension lies in the triceps (quadriceps) muscle."

Potel believed when he first studied the malformation that the muscular contracture was spasmodic in origin, and later[66] that it was due to simple arrest of development of the muscle. I have recently been enabled to observe a case of this relatively rare deformity which, I believe, sheds fresh light upon the cause of the quadriceps contracture.

CASE REPORT

An otherwise healthy female child, aged 5 weeks, was born with a deformity of the right leg. The infant was the firstborn of healthy and unrelated parents, there was no history of deformity occurring in any blood relative, and the pregnancy and labour had been uneventful.

On Examination.—A definite degree of hyperextension at the right knee was evident (*Figs.* 475, 476), with two transverse furrows occupying the angle in front of the joint. The femoral condyles were easily palpable as projections in the popliteal fossa. The thigh appeared slightly wasted, but it was difficult to estimate the degree with accuracy owing to a plenteous deposit of subcutaneous fat.

On attempting to flex the knee an elastic resistance was felt and was clearly due to shortening of the quadriceps extensor apparatus. On releasing the leg the knee-joint at once returned to the hyperextended position. Radiography showed the hip-joints to be normal.

The infant was admitted to the Church of Scotland Deaconess Hospital and manipulative reduction was twice attempted under anæsthesia. On both occasions it was found that by steady pressure flexion to about 90° could be obtained, but subsequent radiography showed

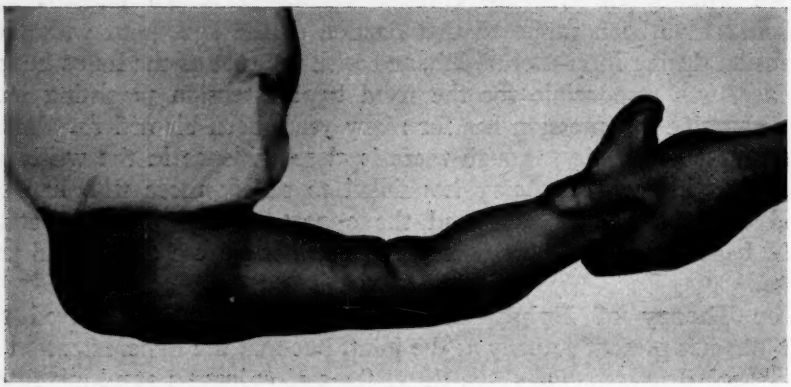

FIG. 475.—Congenital genu recurvatum in a female of 18 months.

FIG. 476.—Radiogram at 18 months showing hyperextension deformity of the knee-joint.

FIG. 477.—Radiogram at the age of 6 weeks to illustrate the pseudo-reduction produced by manipulation. The popliteal surface of the tibia lies in contact with the articular aspect of the femur.

that the reduction was a false one, the popliteal aspect of the tibia lying in contact with the articular aspect of the femoral condyles (*Fig.* 477).

At the age of 18 months the hyperextension deformity was still present, though a striking change in the contour of the tibia had taken place, this bone being curved in its upper third with a decided anterior convexity, the apex of the curve coinciding roughly with

the attachment of the patellar tendon to the tibial tuberosity. This secondarily acquired deformity is of great interest, as it demonstrates the striking effect which abnormal muscular pull may play in the production of bony deformities (*Figs.* 476, 478).

OPERATION.—Operative reduction of the subluxation was now undertaken. A vertical incision was made over the antero-lateral aspect of the knee-joint and was carried well up the thigh. The incision was deepened to expose the quadriceps muscle and the knee-joint. The quadriceps muscle was found to be atrophied, and appeared to the naked eye to be composed almost entirely of fat and fibrous tissue. Division of the quadriceps tendon and of its lateral expansions at once liberated the proximal extremity of the tibia, which slipped, under the influence of gravity alone, into its normal relationship to the condyles of the femur (*Fig.* 478). Several portions of tissue were removed from the quadriceps for microscopical examination and the wound was closed.

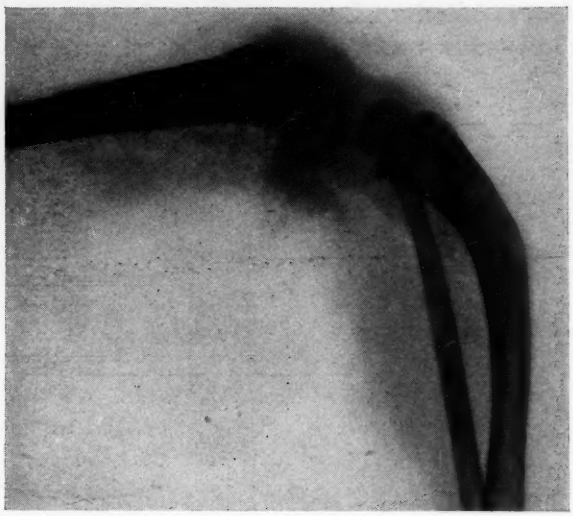

FIG. 478.—Radiogram illustrating the true reduction of the subluxation resulting from open division of the quadriceps tendon. The angling of the tibial diaphysis results from the abnormal traction through the shortened quadriceps muscle.

PATHOLOGY.—Sections of the quadriceps muscle showed small areas of normal muscular tissue embedded in large tracts of fat and fibrous tissue. In some parts very fine atrophied fibres are visible and many normal muscle spindles can be seen standing out from the surrounding fat and fibrous tissue (*Figs.* 479–481). No definite immature fibres at the myoblastic stage of development were recognized, though in places the atrophied fibres had a rather similar appearance.

SUBSEQUENT PROGRESS.—On re-examination when the child was 3 years and 3 months old, it was seen that the deformity remained reduced and a surprising degree of stability existed in the joint, the child, who was learning to walk, being able to bear a considerable amount of weight upon the affected limb.

DISCUSSION

The deformity of congenital genu recurvatum, though frequently, as in the case just described, encountered as a solitary malformation, is nevertheless not uncommon as a part of the form of generalized contractures of joints which has been variously known as 'arthrogryposis multiplex congenita', 'amyoplasia congenita', or 'myodysplasia congenita', and to which I have applied the name 'myodystrophia deformans fœtalis'. In a previous paper[54] I was able to show that the contractures of this deformity were due to an intra-uterine fibro-fatty degeneration of the striated muscle of the limbs. The naked-eye and microscopic changes in the shortened quadriceps muscle of this case of congenital genu recurvatum demonstrate beyond doubt the underlying similarity of the pathological process in such contractures, whether the condition be localized to one or both quadriceps or whether it be a part only of a generalized deformity associated with club-feet, club-hands, congenital dislocation at the hip-joints, and bizarre contractures of other joints.

In the previous paper referred to above I have been able to describe the

pathological changes in the generalized myodystrophy and to state my opinion that the condition is essentially an intra-uterine degeneration of fully or partially

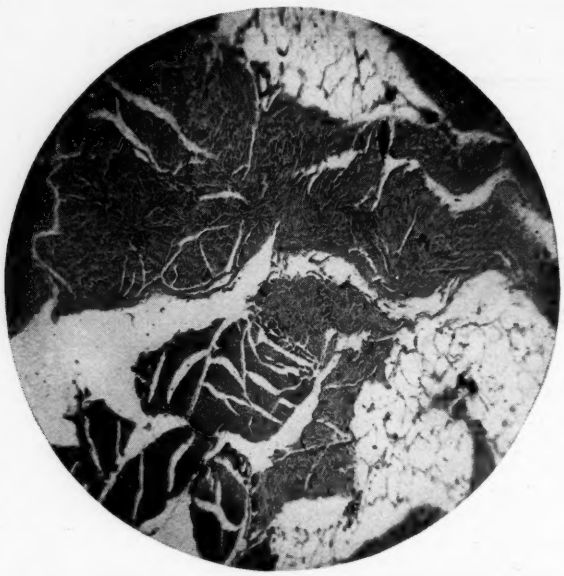

FIG. 479.—Section of quadriceps muscle. The section shows masses of fibrous tissue with patches of fat. (× 45.)

FIG. 480.—Section of quadriceps muscle. Areas of normal muscle tissue are seen to be surrounded by an excess of fibrous tissue. (× 90.)

FIG. 481.—Section of quadriceps muscle. Atrophic muscle fibres and a muscle spindle appear in the lower part of the field, while fat replacement and a nerve-trunk are seen in the upper part. (× 90.)

developed muscle fibres rather than a simple failure of muscle to develop. I have also concluded for various reasons that the lesion is a peripheral one and that the

muscular degeneration is not secondary to an embryonic lesion of the nervous system. In this connection it is interesting to note that the degenerated muscles contain their full quota of nerve-trunks and that the presence of many undegenerated muscle spindles, as found in the muscular dystrophies of post-natal life, is a strong argument in favour of a normal sensory supply to the muscle. Finally, Sherrington[80] has demonstrated by examination of an amyelous fœtus that full and normal development of the striated muscles can take place in the complete absence of motor nerve-supply.

SUMMARY

1. A fresh case of congenital genu recurvatum is described along with naked-eye and microscopic observations.

2. A consideration of these findings makes it clear that the deformity is caused by contracture of the quadriceps extensor cruris muscle resulting from intra-uterine fibro-fatty degeneration of the striated muscle fibres.

3. The pathological process is in all respects similar to that described in myodystrophia deformans fœtalis.

BIBLIOGRAPHY

[1] ARDOUIN, P., *Rev. d'Orthop.*, 1907, viii, 193.
[2] BACILIERI, L., *Arch. f. orthop. u. Unfall-Chir.*, 1905, iii, 213.
[3] BAKER, H. F., *Lancet*, 1881, ii, 951.
[4] BALDWIN, C. H., *Jour. Bone and Joint Surg.*, 1926, viii, 822.
[5] BARTH, A., *Arch. f. klin. Chir.*, 1885, xxxi, 670.
[6] BARWELL, R., *Proc. Roy. Med. and Chir. Soc.*, 1875–80, viii, 218.
[7] BAZERT, L., *Rev. d'Orthop.*, 1925, xii, 155.
[8] BERTIN, J., *L'Union méd.*, 1880, xxx, 616.
[9] BISPING, *Zeits. f. orthop. Chir.*, 1903, xi, 880.
[10] BLANC, E., *Gaz. méd. de Paris*, 1884, i, 325.
[11] BOORSTEIN, S. W., *Med. Jour. and Record*, 1926, cxxiv, 541.
[12] BOORSTEIN, S. W., *Amer. Jour. Child. Dis.*, 1929, xxxviii, 107.
[13] BOULARAN and BOUNHOURE, *Rev. d'Orthop.*, 1923, x, 245.
[14] BRODHURST, B. E., *On the Nature and Treatment of Clubfoot*, 1856, 32. London.
[15] CHEYNE, W., *Lancet*, 1890, ii, 925.
[16] CONRAD, *Corr.-Blatt, f. schweiz. Aertze*, 1875, v, 153.
[17] CORNET, J., *Arch. franco-belges de Chir.*, 1927, xxx, 536.
[18] CURTILLET, J., and LOMBARD, P., *Rev. d'Orthop.*, 1912, iii, 289.
[19] DELANGLADE, E., *Ibid.*, 1903, iv, 193.
[20] DEROCQUE, P., *Ibid.*, 54.
[21] DOWD, C. N., *Ann. of Surg.*, 1899, xxix, 345.
[22] DREHMANN, G., *Zeits. f. orthop. Chir.*, 1900, vii, 459.
[23] FRANCINI, M., *Chir. d. Org. di Movimento*, 1917, i, 268.
[24] FRASER, J., *Surgery of Childhood*, 1926, ii, 1105. London.
[25] GIBNEY, V. P., *Ann. of Surg.*, 1899, xxix, 346.
[26] GODLEE, R., *Lancet*, 1877, i, 316.
[27] GRISWOLD, A. S., *Jour. Bone and Joint Surg.*, 1927, ix, 628.
[28] GROSSMANN, J., *Med. Jour. and Record*, 1927, cxxv, 189.
[29] GUENIOT, *Bull. et Mém. Soc. de Chir.*, 1880, vi, 442.
[30] HABS, *Münch. med. Woch.*, 1905, xii, 556.
[31] HAMADA, G., *Brit. Med. Jour.*, 1930, ii, 141.
[32] HAMILTON, *Treatise on Fractures and Dislocations*, 1891, 8th ed., 838. London.
[33] HENRARD, E., *Monats. f. Geburtsh. u. Gynäkol.*, 1928, lxxx, 317.
[34] HOLTZMANN, H., *Virchow's Arch.*, 1895, clx, 272.
[35] HORVÁTH M., *Wien. med. Woch.*, 1897, xlvii, 1623.
[36] JAGERINK, *Nederl. Tijds. v. Geneesk.*, 1927, ii, 476.
[37] JOACHIMSTAL, *Berl. klin. Woch.*, 1889, 923.

[38] KAREWSKI, *Arch. f. Kinderheilk.*, 1891, xii, 234.
[39] KIRMISSON, E., *Rev. d'Orthop.*, 1903, iv, 413.
[40] KNAUER, E., *Monats. f. Geburtsh. u. Gynäkol.*, 1897, v, 1.
[41] KOFMANN, S., *Arch. f. orthop. u. Unfall-Chir.*, 1907, vi, 41.
[42] KOFMANN, S., *Jour. Bone and Joint Surg.*, 1930, xii, 871.
[43] KÖNIG, *Berl. klin. Woch.*, 1898, xxxv, 496.
[44] KUH, *Deut. med. Woch.*, 1910, xxxvi, 1783.
[45] KÜSTNER, O., *Arch. f. klin. Chir.*, 1880, xxv, 601.
[46] LIPSCOMB, T. W., *Austral. Med. Gaz.*, 1903, xxii, 568.
[47] LITTLE, W. J., *On the Nature and Treatment of the Deformities of the Human Frame*, 1853, 319. London.
[48] MAAS, H., *Arch. f. klin. Chir.*, 1874, xvii, 492.
[49] MCFARLAND, B. L., *Jour. Bone and Joint Surg.*, 1929, xi, 281.
[50] MCGILLICUDDY, T. J., *Jour. Amer. Med. Assoc.*, 1892, xix, 106.
[51] MAGNUS, F., *Deut. Zeits. f. Chir.*, 1905, lxxviii, 555.
[52] MASON, *Med. Record*, 1877, xii, 42.
[53] MAYER, L., *Amer. Jour. Orthop. Surg.*, 1912–13, x, 411.
[54] MIDDLETON, D. STEWART, *Edin. Med. Jour.*, 1934, xli, 401.
[55] MOUCHET, A., *Bull. et Mém. Soc. anat. de Paris*, 1904, lxxix, 856.
[56] MUSKAT, G., *Arch. f. klin. Chir.*, 1897, liv, 852.
[57] MUTEL, *Rev. d'Orthop.*, 1911, ii, 302.
[58] MYERS, T. H., *New York Med. Jour.*, 1890, li, 444.
[59] OLIVIERAS, D., and BONREPAUX, J., *Rev. méd. de Barcelona*, 1924, i, 28.
[60] OWEN, E., *Lancet*, 1891, i, 989.
[61] PÉRIER, E., *Bull. et Mém. Soc. de Chir.*, 1880, vi, 682.
[62] PHOCAS, G., *Rev. d'Orthop.*, 1891, ii, 50.
[63] POST, G. E., *Med. Record*, xiii, 408.
[64] POTEL, G., "Etude sur les Malformations congénitales du Genou", *Thèse de Lille*, 1897.
[65] POTEL, G., *Echo Méd. du Nord*, 1897, i, 372.
[66] POTEL, G., *Traité pratique d'Orthopédie*, 1925, 186. Paris.
[67] PRINCE, L. D., *Surg. Gynecol. and Obst.*, 1917, xxiv, 714.
[68] REINER, M., *Zeits. f. orthop. Chir.*, 1904, xiii, 442.
[69] RICHARDSON, W. L., and PORTER, C. B., *Boston Med. and Surg. Jour.*, 1875, xcii, 321.
[70] RIDLON, J., *Trans. Amer. Orthop. Assoc.*, 1896, ix, 179.
[71] ROBERTS, J. B., *Ann. of Surg.*, 1901, xxxiv, 281.
[72] ROBERTS, J. B., *Ibid.*, 1909, xlix, 276.
[73] ROBERTSON, J. K., *Glasgow Med. Jour.*, 1884, xxii, 118.
[74] ROBIN, *Rev. d'Orthop.*, 1914–17, v, 237.
[75] ROSENFELD, L., *Zeits. f. orthop. Chir.*, 1902, x, 405.
[76] SANDERS, A., *Monats. f. Geburtsh. u. Gynäkol.*, 1928, lxxix, 68.
[77] SAYRE, L. A., *Rev. mens. d. Mal. d. l'Enf.*, 1890, viii, 453.
[78] SELLS, H., *Brit. Med. Jour.*, 1883, i, 766.
[79] SHATTOCK, S. G., *Trans. Pathol. Soc. Lond.*, 1891, xlii, 280.
[80] SHERRINGTON, C. S., *Jour. of Physiol.*, 1894–5, xvii, 211.
[81] SIMPSON, G., *Brit. Med. Jour.*, 1893, ii, 1099.
[82] SKELTON, E. W., *Long Island Med. Jour.*, 1910, iv, 24.
[83] SORREL, E., and MME. LE GRAND-LAMBLING, *Bull. Soc. Péd.*, 1932, xxx, 169.
[84] SPEIRS, H. W., *Jour. Bone and Joint Surg.*, 1927, ix, 469.
[85] STEELE, A. J., *Trans. Amer. Orthop. Assoc.*, 1896, ix, 175.
[86] TARNIER, *Bull. et. Mém. Soc. anat. de Paris*, 1854, 109.
[87] TAYLOR, H. L., *Trans. Amer. Orthop. Assoc.*, 1895, viii, 280.
[88] TIMMER, H., *Nederl. Tijds. v. Geneesk.*, 1892, xxviii, 778.
[89] TRIDON, P., *Rev. d'Orthop.* 1905, vi, 497.
[90] TSCHARNOMSKAJA, J. J., *Zentralb. f. Chir.*, 1902, xxix, 791 (Abstract).
[91] VON SALIS, H., *Deut. Zeits. f. Chir.*, 1908, xciv, 149.
[92] WEHSARG, R., *Arch. f. orthop. u. Unfall-Chir.*, 1905, iii, 197.
[93] WENTWORTH, E. T., *Jour. Bone and Joint Surg.*, 1928, x, 585.
[94] WITTEK, A., *Deut. Zeits. f. Chir.*, 1930, ccxxv, 308.
[95] WOLFF, J., *Zeits. f. orthop. Chir.*, 1893, ii, 23.
[96] WUTZER, *Arch. f. Anat. u. Phys.* (Müller), Berlin, 1835, 385.

OBSERVATIONS ON FISTULA IN ANO IN RELATION TO PERIANAL INTRAMUSCULAR GLANDS

WITH REPORTS ON THREE CASES

By Sir CHARLES GORDON-WATSON and HAROLD DODD, London

It was pointed out as far back as 1880 by Herrmann and Desfosses[1] that some cases of fistula in ano might be caused by glands penetrating deeply from the lower rectal mucous membrane into the perianal tissues: "Au point de vue chirurgical ces longs conduits tortueux coiffés à leur extrémité par de petits follicules clos, présentent un certain intérêt, en regard notamment au rôle qu'ils peuvent jouer dans la production des fistules compliquées qu'on rencontre si fréquemment dans cette région." The same authors showed that the glands reached and actually invaded the muscle of the internal sphincter after passing through the muscularis mucosæ.

In 1929 Lockhart-Mummery, in an address on fistula in ano at the Royal Society of Medicine,[2] referred to Cuthbert Dukes's statement that intramuscular glands can often be seen in complete sections of the anal region, and to his suggestion that they might be a possible cause of fistulæ. He pointed out that these glands had, until comparatively recently, been apparently overlooked by anatomists and surgeons.

ANATOMY AND DEVELOPMENT OF PERIANAL INTRAMUSCULAR GLANDS

These glands are variable in number and distribution, and seem to serve no particular purpose. It has been suggested that they are the remains of odoriferous glands, which may have played an important rôle in sex attraction before our ancestors assumed the erect posture. *Fig.* 482 illustrates these glands and their relation to the anal canal.

Further interesting details explaining the development of these glands were given by Professor H. A. Harris[3] when he spoke on "Some Embryological Aspects of the Problem Involved" in the above discussion on fistula in ano. Harris pointed out that when parent tubes give rise to daughter tubes—such as the œsophagus to the trachea and to the bronchial tree; the duodenum to the pancreas, to the liver, and to the biliary system; and the rectum to the urogenital sinus—then irregularities in the muscular and epithelial layers of the original tubes are not infrequent. Atresia, stenosis, and cyst formation may occur, whilst the muscular layer, which is weakened by the outgrowth and retarded in development, lends itself to diverticulosis.

Harris also emphasized that certain of the intramuscular glands grow deeply to reach the inner circular layer before the muscularis mucosæ is differentiated.

Histologically the glands are lined by transitional epithelium from the epithelium of the anal canal at the anorectal junction, and the structure is that of

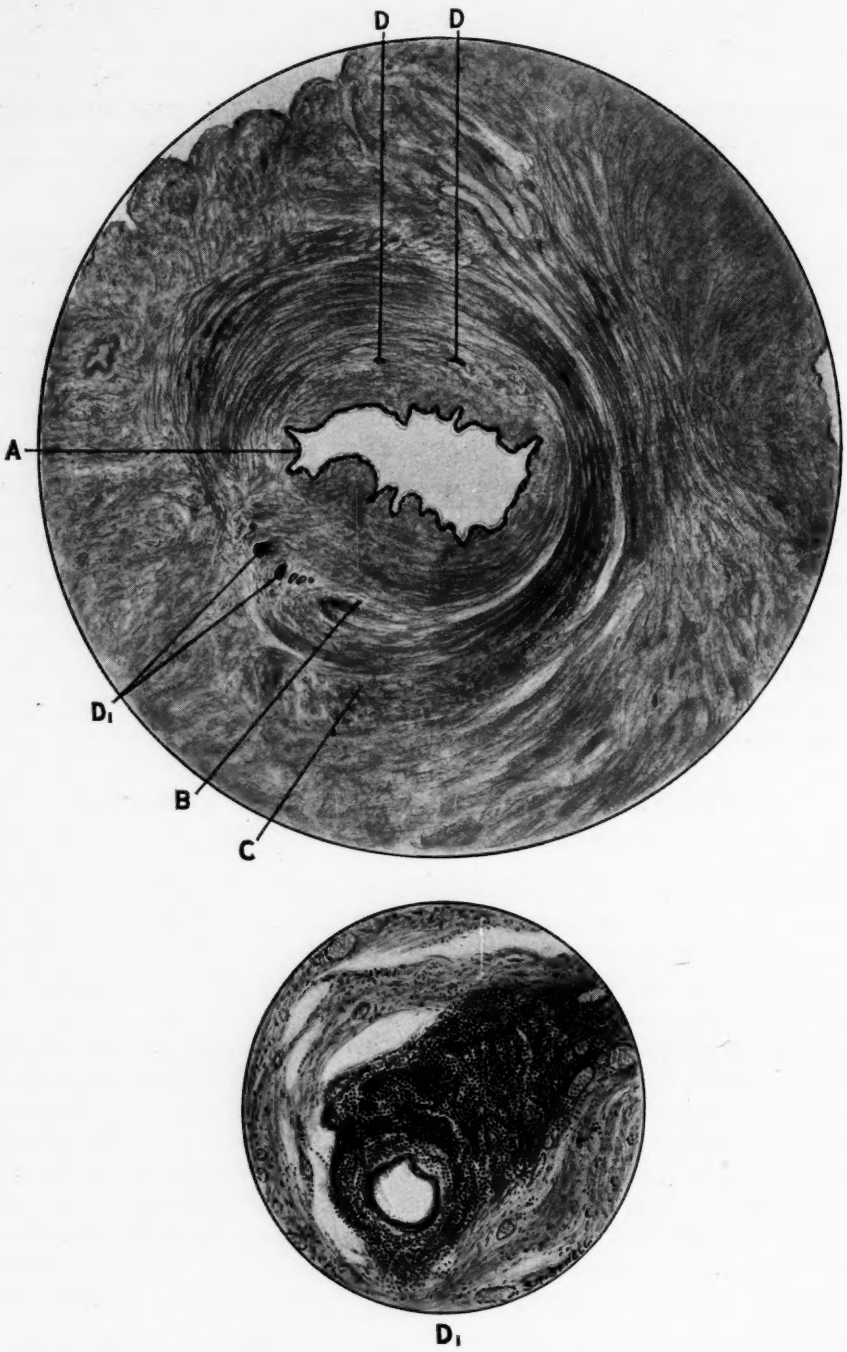

FIG. 482.—Transverse section of the anal canal. A, Stratified epithelial lining B, Internal sphincter; C, Tendon of longitudinal muscle of rectum surrounded by the external sphincter; D, Intramuscular glands buried in the substance of the internal sphincter muscle. D_1 (inset), Higher magnification of an intramuscular gland surrounded by round-celled infiltration. (*From St. Mark's Hospital Museum.*)

either convoluted mucous or of sweat glands. The glands grow outwards into the loose tissue within the internal sphincter and into the limiting annulus of connective tissue which separates the internal sphincter above from the external sphincter below. Before the muscularis mucosæ is developed the glands may arrive at and actually penetrate the internal sphincter and the external longitudinal coat, and even spread: (1) To the superficial surface of the levator ani and so into the ischiorectal fossa; (2) Deep to the levator ani into the true pelvis; (3) Into the substance of the levator ani muscle.

In post-natal life these glands may be recognized deep to the internal sphincter, in the substance of the external sphincter, or even in the levator ani.[3]

THE RELATIONSHIP OF ANORECTAL GLANDS TO FISTULA IN ANO

Having established the presence, development, and position of deep intramuscular perianal glands, it is not difficult to appreciate that pathological changes may readily occur in them.

It seems probable that these glands are, for the most part, vestigial and often lose connection with the anal canal by obliteration of the ducts during development, and that in some instances when the duct is patent infection from the anal canal takes place, as is illustrated by the three cases recorded below. In other instances infection may occur in the glandular substance when there is no direct communication with the anal canal, or when the orifices of the ducts have become blocked by congestion of the mucosa and submucosa.

If an abscess occurs in one of these glands, it is easy to visualize the onset of a perianal or ischiorectal abscess and a subsequent fistula. On the rarer occasions when one of these glands has penetrated to the deep surface of the levator ani, we may have an explanation for the development of a pelvirectal abscess and fistula.

The presence of these deep glands emphasizes the importance of a careful search for a rectal opening when dealing with a supposed blind external fistula. When such a track is overlooked, permanent relief cannot be effected, and the fistula will not be cured.

In the past it has been a little difficult to explain why it happens that so many fistulæ in ano appear to have been chronic from the first and have failed to present any initial history of an acute onset or of abscess formation. It now seems quite easy to believe that a chronic origin may well be the truth in some cases, should a mild infection of a perianal gland occur and the existence of a patent duct permit drainage into the anal canal. In other instances in the absence of a patent duct it is equally easy to believe that infection of the perianal glands may well be responsible for many of the acute or subacute abscesses which arise for no apparent reason.

Unless tuberculous disease or malignant invasion of a chronic fistula is suspected, it is not the usual practice to excise fistulous tracks, and submit them to histological examination; so that it is, perhaps, not surprising that the observations recorded below appear to have escaped recognition in the past.

The existence of these perianal glands, often with patent ducts leading into the rectum; the tuberculous origin of many fistulæ; and the fact that malignant

changes sometimes occur in fistulous tracks combine to emphasize the importance of a histological examination when operating for fistula in ano.

Obviously it can happen only occasionally, and then in the case of a recently formed abscess or fistula, that the epithelium of these glands and their ducts lining either abscess or fistula is recognized histologically. Once such a case has become chronic, granulation tissue will have replaced the epithelium and the real origin becomes obscured.

Although the theory that these deep perianal glands might cause an abscess or fistula was tentatively suggested as far back as 1880, and has been revived more recently, no clinical and histological evidence appears to have been brought forward to support this view until one of us (C. G.-W.) in January, 1932, before the Subsection of Proctology at the Royal Society of Medicine, recorded a case which seemed quite clearly to support the above views (*Case* 1 below). Since then two other similar cases have been met with and are now reported. These cases with histological illustration are, in all probability, the first to be recorded, at any rate in this country. It is hoped that the records will stimulate others to confirm the relationship between some of the fistulæ in ano and the intramuscular glands.

CASE REPORTS

Case 1.—An Oxford undergraduate, aged 21, came under the care of one of us (C. G.-W.) on Dec. 9, 1931, complaining of pain in the region of the anus for the past three days.

ON EXAMINATION.—There was a small localized inflammatory swelling about ¾ in. from the anal margin on the right side, which was recognized as an early ischiorectal abscess.

OPERATION.—At operation the anal canal was examined for evidence of any communication between the abscess and the anal canal. On squeezing the abscess area a little pus exuded from beneath one of the valves of Morgagni. A director was passed through this opening into the abscess cavity and the abscess incised through the skin. The track leading from the abscess to the anal canal passed through the substance of the external sphincter. The lining of this track was carefully dissected out and the radical operation completed. (*Fig.* 483.)

PATHOLOGICAL REPORT.—The following is a copy of the report made by Dr. Cuthbert Dukes on the specimen sent to him: The fistulous track has a complete lining of a transitional type of epithelium, mostly from three to six cells in depth. The subepithelial tissues are infiltrated with inflammatory cells, chiefly small and large mononuclear cells. There are also some collections of inflammatory cells in the muscle and connective tissue surrounding the fistula, but the histology is not in any way suggestive of a tuberculous lesion.

Comment by Dr. Dukes.—Fistulous tracks are rarely lined with epithelium. The congenital sacrococcygeal fistula is lined by stratified epithelium with rudimentary sebaceous glands and hair follicles, none of which are found in this case. A long-standing septic fistula may have a well-developed lining of stratified epithelium due to the skin growing in from the edge, but the epithelium lining this fistula has not these histological appearances. The track of this fistula closely resembles a duct of one of the intramuscular glands which pass from the upper part of the anal canal into the sphincter muscles.

Case 2.—A man (C. E. D.), aged 48, was under the care of one of us (H. D.) in May, 1932. In January, 1932, the patient noticed a painless swelling to the right of the anal margin. By February the swelling had spread, was painful, and after fomentations burst and discharged a small amount of pus, which continued until he was first seen.

On May 23 he was admitted to hospital and a small external fistula was found about 1½ in. from the anus at 11 o'clock. No induration was detected between the rectum and the fistula, but pressure on the area exuded a small bead of thin pus.

OPERATION (May 26).—Under low spinal anæsthesia the fistula was laid open on a director and a granulating surface ending in a small abscess cavity was revealed. The area involved, together with the surrounding skin, was excised *en masse*. On cleaning the wound a thin bead of pus exuded from the centre of its soft fatty surface. This area was explored

FIG. 483.—*Case* 1. Illustrating a fistula arising from an intramuscular gland. A, Section of the track of an acute ischiorectal abscess passing through the sphincter into the anal canal. The track is lined with epithelium similar to that of the intramuscular glands. (× 16.) B, Higher magnification (× 100).

with a fine probe and a track found leading through the lower fibres of the external sphincter into the rectum. The track was excised.

After operation the excised pieces were threaded on silkworm gut, and the epithelial-lined track could be seen running directly into the abscess cavity (*Fig.* 484). After preservation in formalin the duct showed up even more clearly.

PATHOLOGICAL REPORT.—Dr. Cuthbert Dukes reports on the excised pieces as follows:—

1. *Region of External Opening.*—In this region the fistulous track is lined with granulation tissue of the type found in infections with pyogenic bacteria and there is no sign of an epithelial lining.

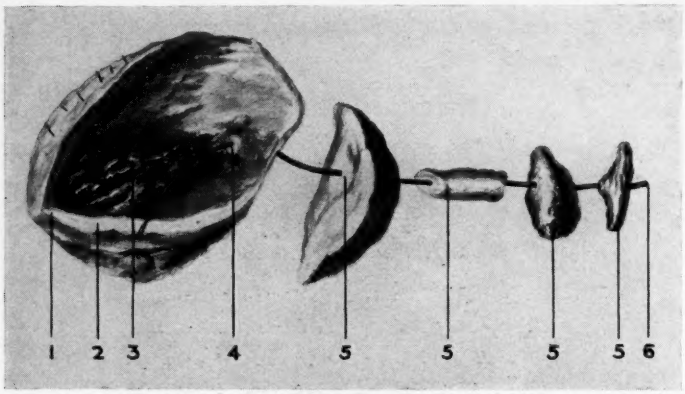

FIG. 484.—*Case* 2. The fistulous track dissected out to the rectum. 1, The external orifice of the fistula; 2, The skin of the buttock surrounding 1; 3, The granulating base of the fistulous track; 4, The orifice of the epithelial-lined track in 3; 5, The track running through to the rectum; 6, A piece of silk-worm gut.

2. *Deeper Portion of Track.*—In this region the fistula is lined with a complete layer of stratified epithelium containing neither sebaceous nor sweat glands nor hair follicles. Interspersed in the surrounding tissues are a few collections of inflammatory cells with some giant cells amongst them. Another piece of tissue from the same region includes only a portion of the fistula, but shows the same features.

I think a possible explanation is that this fistula arose from one of the intramuscular glands which open into the lower part of the anal canal—that is to say, below the level of the valves.

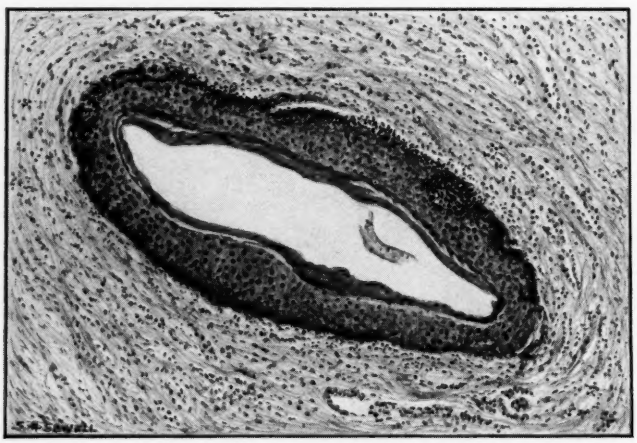

FIG. 485.—*Case* 2. Microscopic section of the epithelialized track. (× 40.)

This portion of the anal canal is covered with stratified epithelium not containing any sweat glands or sebaceous glands or hair follicles. The track of the fistula has a similar lining (*Fig.* 485). The fact that there is no evidence of any epithelium at the external opening proves that this is not a simple case of epithelialization of the track of a chronic fistula.

Case 3.—A man (C. G.), aged 36, under the care of one of us (C. G.-W.) was admitted to hospital with the following history: Six months previously he began to be troubled with piles. Four months previously he first noticed some yellowish discharge from the anus. Recently there has been some pain and tenderness at the anal margin.

ON EXAMINATION.—On rectal examination a small perianal swelling was detected at the anal margin anteriorly, about the size of a pea. The swelling was firm and appeared to be in the substance of the sphincter. On examination of the anal canal a small fistulous track, about $\frac{1}{2}$ in. long, was detected leading directly into the swelling.

OPERATION (June 18, 1934).—A probe was passed along the track into the abscess cavity and the abscess cavity dissected out intact from the substance of the external sphincter, and then a radical operation for direct fistula was carried out. Some hæmorrhoids were also ligatured. Recovery was straightforward.

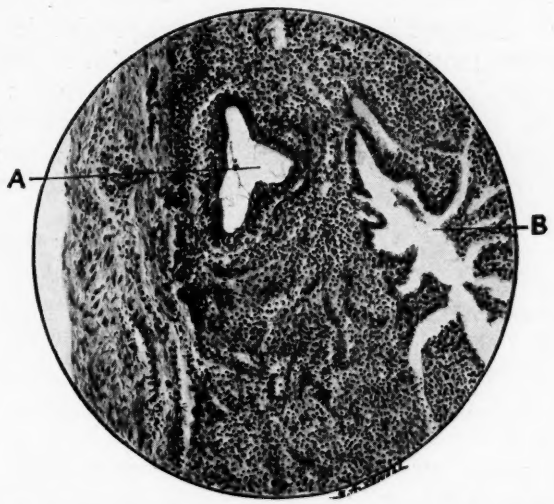

FIG. 486.—*Case* 3. Abscess surrounding intramuscular gland. The section passes transversely across two terminal branches of an intramuscular gland. In the branch marked A the epithelial lining is intact, but in B only the upper half of the lumen is covered by epithelium, and the appearances suggest that the duct is opening directly into the abscess. (× 70.)

PATHOLOGICAL REPORT.—Dr. Cuthbert Dukes reported as follows:—

Tissue from Anal Region.—The fragment consists of sphincter muscle and connective tissue from the anal region. A small abscess is situated between the fibres of the sphincter muscle, and in contact with this is the duct of a gland lined by transitional epithelium. This duct has the histology of an intramuscular gland arising from the anal canal, and the appearances suggest that the abscess is related to the presence of the gland. (*Fig.* 486.)

SUMMARY

1. Some observations are made on deep intramuscular perianal glands in relation to the cause of abscess and fistula in ano.

2. Three cases of fistula in ano lined with glandular epithelium are recorded.

3. Microphotographs of each case are submitted.

REFERENCES

[1] HERRMANN, G., and DESFOSSES, L., *Comptes rend. Acad. des Sci.*, 1880, xc, 131.
[2] LOCKHART-MUMMERY, J. P., *Proc. Roy. Soc. Med.*, 1929, xxii, 1331.
[3] HARRIS, H. A., *Ibid.*, 1341.

SCHIMMELBUSCH'S DISEASE OF THE BREAST AND DR. A. LACASSAGNE'S EXPERIMENTS ON MICE

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The development of carcinoma in the breasts of young male mice after the injection of œstrin is one of the most interesting experiments Dr. Lacassagne has recently performed. I am very grateful to him for having shown me his microscopical sections and for his courtesy in allowing me to reproduce them before he has published them himself. The object in writing this article is to point out that the morphological appearances seen in these sections are the same as those in all the stages of the process that I have described as Schimmelbusch's disease when it ends in carcinoma. The similarity of my description of this disease and the experimental changes that occur in the breasts of his mice had already struck Dr. Lacassagne before I had seen his sections and discussed the matter with him.

My conception of Schimmelbusch's disease differs from Schimmelbusch's description only in one respect. To his description I add the formation of cysts as an essential preliminary stage to the procession of events that may end in carcinoma. Otherwise, with minor exceptions, I agree entirely with his masterly treatise, and see no reason why his name should be omitted from the nomenclature of the disease he described.

With my addition, Schimmelbusch's disease begins in a desquamative epithelial hyperplasia, sometimes in the ducts only, and sometimes in ducts and their acini. The disease may affect one duct only, or only one duct and its acini. It may be more widely distributed. The affected ducts are dilated by the presence of colostrum-like cells in a fluid medium. The end of this stage is the formation of duct and acinous cysts. It begins in the late twenties or early thirties, and may last an indefinite time or pass on to its next stage at once. It is interesting to quote at this point an observation Dr. Lacassagne has made on the contents of these cysts. He says: "Enfin nous avons commencé à rechercher l'hormone dans le tissue mammaire de seins prélevés chirurgicalement. Dans le cas d'une Femme de 57 ans un épithélioma occupait une portion de la glande; des sections profondes, dans la partie non cancéreuse de l'organe, ont permis de faire sourdre un liquide provenant de l'ouverture de petits kystes. Ce liquide, injecté à la dose de 1 c.c., a provoqué l'œstrus. En revanche, un broyat du tissu cancéreux lui-même s'est montré inactif. . . ." And in the paper in which he made this statement he concludes: "Le colostrum contient normalement une quantité certainement très élevée de folliculine. Cette hormone peut également être trouvée dans la glande mammaire de Femmes atteintes de cancer du sein."

If the process proceeds at once, the next stage—which really is the beginning of Schimmelbusch's description—is the development of multiple epithelial neoplastic growths within the cysts. These tumours do not transgress their dilated normal

boundaries. They may or may not be papillomatous in character, although they very often are. They may be limited only to the ducts, or they may also grow in the acini. I have not seen them only in the acini.

This second stage usually occurs in the late thirties or early forties. The tumours may remain within their normal boundaries for an indefinite time, although they continue to grow in size and increase in number. If the process continues, the next and final stage of carcinoma begins in the late forties and early fifties. The time of appearance of these three stages is not always so stable as their sequence. For example, Mr. Geoffrey Keynes has recently allowed me to cut whole microscopical sections of a breast he removed from a woman aged 27 years. According to my conception of them, the disease had already passed through the cystic and benign neoplastic stages, and reached the carcinomatous end—all of which stages are to be seen in the sections.

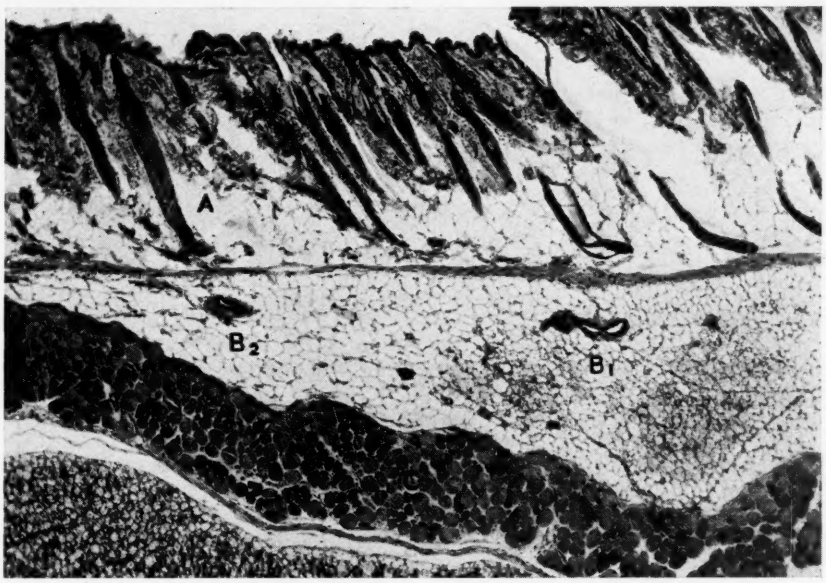

FIG. 487.—Photograph of the normal and immature breast of a male mouse. A, Skin; B_1 and B_2, Two ducts; C, Muscle fibres.

Turning now to Dr. Lacassagne's experiments on young male mice. He injected œstrin into their bodies and cut microscopical sections of their breasts in consecutive stages of their treatment. He used mice belonging to carcinoma strains and also mice in which no such strain was known to exist. In the breasts of the latter carcinoma developed later than in those of the former. Dr. Lacassagne states that the adeno-carcinomata following the administration of œstrin behave like the disease that arises spontaneously in mice. Metastases occur with great frequency and generally in the lungs. At this point the reader should study the photographs of Dr. Lacassagne's sections (*Figs.* 487–493).

After studying these sections the reader will be struck by several sets of impressions, to some of which I now draw attention. It will be fairly obvious that the changes in the newly-formed breast tissue are the same in kind and sequence as in Schimmelbusch's disease. It also appears obvious that the changes leading

to carcinoma do not occur in the normal immature breasts of the mice, but that they appear in the new and abnormal breast tissue. Considering the size of the animals, the cysts are large ones. They resemble the duct cysts of Schimmelbusch's disease in that they do not coalesce to form larger ones. The sections support my contention that Schimmelbusch's disease is a disease *sui generis* and unrelated to

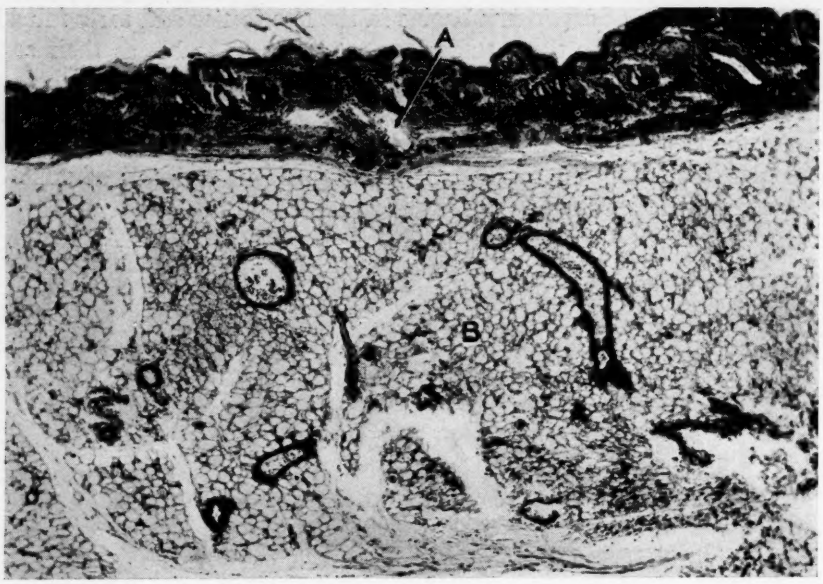

FIG. 488.—Photograph of the breast of a male mouse after forty-eight days' treatment with œstrin. A, Skin; B, A layer of fat in which can be seen five or six ducts. There is a slight increase of breast tissue, the dilated ducts of which contain desquamated epithelial cells.

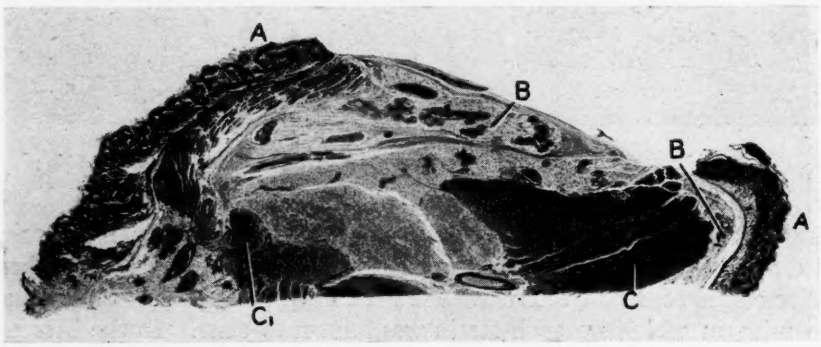

FIG. 489.—Low-power photograph of a whole section of the breast of a male mouse after eighty-five days' treatment with œstrin. A, Skin; B, Breast tissue that shows a decided increase in amount, and a state of multiple cysts, most of which appear to be formed by dilated ducts; C and C_1, Musculature. (*Cf. Fig.* 490.)

mazoplasia (so-called chronic mastitis), of which there is not the slightest sign. They also support my belief that all the cystic breasts in Schimmelbusch's disease should be removed. In carrying out this practice for some years I have met with the following results in the total excision of these breasts: (1) Some of them have been only cystic; (2) Others contained cysts with papillomatous and other growths

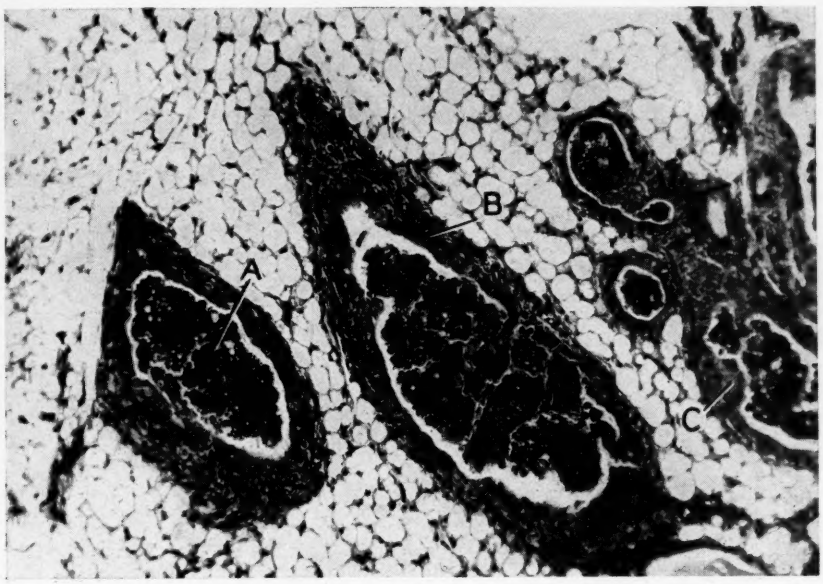

FIG. 490.—High-power photograph of breast tissue from *Fig.* 489 (eighty-five days). Epithelial neoplastic cells (among which many mitoses can be seen) have formed round the margins of what are apparently ducts (A, B, C).

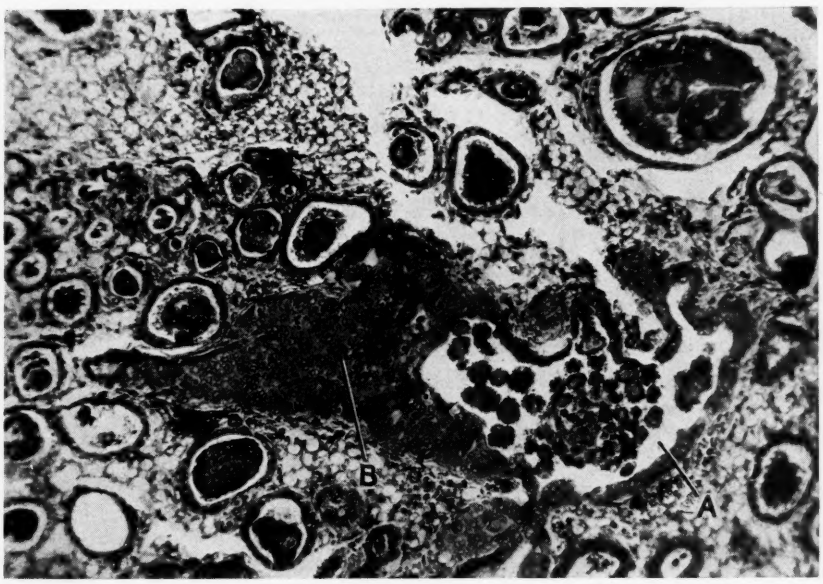

FIG. 491.—Photograph of a part of the breast of a male mouse, 143 days after treatment with œstrin. There has been an enormous increase of breast tissue, which is in a multiple cystic state. At A is apparently a duct in which epithelial neoplastic cells (B) have developed and apparently are still contained within their normal but dilated boundary. There are many mitoses visible in the epithelial neoplasia.

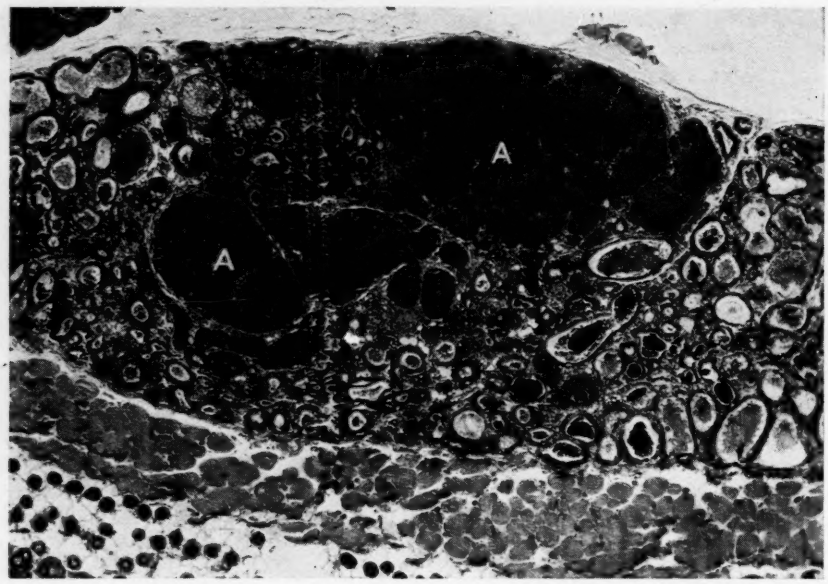

FIG. 492.—Photograph of part of the breast of a male mouse 179 days after treatment with œstrin. The section shows the enormous increase of breast tissue as in the breast of *Fig.* 491, also its multiple cystic state. At A is a well-developed carcinoma. (*Cf. Fig.* 493.)

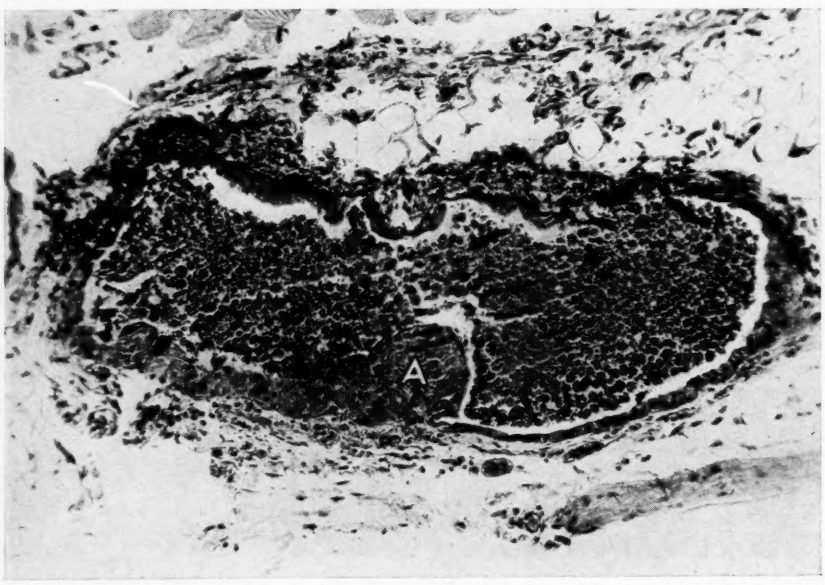

FIG. 493.—Photograph of apparently a duct from a part of *Fig.* 492 far remote from the area of carcinoma. Epithelial neoplasia (A) has occurred that is still confined within its normal but dilated boundary.

confined within their normal boundaries; (3) Others contained definite and unsuspected early carcinoma; and (4) Finally I have met with other instances from which a cyst or cysts had been very locally excised and had subsequently developed carcinoma.

Unfortunately one man's experience is insufficient to provide enough material to form an impressive notion of the difference in numerical relationship between these four conditions. My own experience is that the conditions (1) and (2) are more frequent than (3) and (4), my impression being that (2) is more frequent than (1). Conclusions upon this matter are of no value whatever when they are based only on a partial examination. The only valuable evidence is that based upon the examination of microscopical sections made of the whole mammary glands.

The remaining practical issue of these experiments is as follows :—

Are patients suffering from mazoplasia running any risk by the administration of ' ovarian residue ', i.e., ovarian extract deprived of its luteal contents ? It would be a pity if its use in this condition were contra-indicated, because it so often relieves the symptoms. I cannot conceive that it is dangerous. In considering this point it must be realized that every pregnant woman and mare is flooded with œstrin with no bad effect. It may well be that the oestrin may make matters worse when carcinoma exists in the breast of a pregnant woman, and may be the cause of the particularly malignant type of the disease which is popularly supposed to exist when it occurs in pregnancy.

In all my sections I cannot discover an indication that mazoplasia is related to carcinoma. These sections seem to me to show that it would be unwise to administer ' ovarian residue ' in women suffering from cystic breasts.

BIBLIOGRAPHY

LACASSAGNE, A., " A Propos d'une Pathogénie de l'Adéno-carcinome mammaire : Recherche de la Folliculine dans le Colostrum ", *Comptes rend. Soc. de Biol.*, 1934, cxvi, June, 844.

LACASSAGNE, A., " Sur la Pathogenie de l'Adéno-carcinome mammaire de la Souris ", *Ibid.*, cxv, March 3, 937.

LACASSAGNE, A., " Influence d'un Facteur familial sur la Production, par la Folliculine, de Cancers mammaires chez la Souris mâle ", *Ibid.*, 1933, cxiv, Oct. 21, 427.

LACASSAGNE, A., " Apparition de Cancers de la Mamelle chez la Souris mâle, soumise à des Injections de Folliculine ", *Comptes rend. Acad. des Sci.*, 1932, cxcv, Oct. 10, 630.

SCHIMMELBUSCH, C., *Arch. f. klin. Chir.*, 1892, xliv, 117.

CHEATLE, G. L., and CUTLER, MAX, *Tumours of the Breast*, 1931, 92–159.

RECLUS, P., *Bull. et Mém. Soc. anat. de Paris*, 1883, lxviii, 428.

WHEELER, SIR W. I. DE C., " Breast Tumours ", *Med. Annual*, 1931, 79.

GOORMAGHTIGH, N., and AMERLINCK, A., " Réalisation expérimentale de la Maladie de Reclus de la Mamelle chez la Souis ", *Bull. Assoc. fran. pour l'Etude du Cancer*, 1930, xix, No. 7, July.

OSTEO-ARTHRITIS OF THE HIP-JOINT

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THE condition usually described as osteo-arthritis of the hip-joint is essentially a disease of middle and later life. The very greatest ingenuity has been shown in its classification, and each new investigator feels the urge for further subdivision, until in many of these classifications it is almost impossible to recognize what is after all a very common condition. Fortunately the classification which is generally accepted at present is probably the simplest of all. In it the condition is divided into two main groups: (1) The infective or toxic; and (2) The traumatic. From the fact that two such widely differing causes may produce conditions in the joint which are apparently similar it would seem that osteo-arthritis of the hip is not a definite disease of itself, but is rather the reaction of the joint and articular cartilage to adverse conditions which lead to a loss of their vitality, or to an alteration of the line of weight-bearing through the joint surfaces and must cause an abnormally increased pressure over a restricted area.

FIG. 494.—A case of osteo-arthritis of hip, the result of Perthes' disease in early childhood.

Age Incidence.—A study of 89 cases of osteo-arthritis of the hip, both unilateral and bilateral, on which this paper has been based, reveals some very interesting points in regard to the age of onset of the first symptoms of trouble. In the bilateral cases the average age of onset was 53 years, while in the unilateral group it was 34 years. In calculating these two figures it was found that in a large proportion of the unilateral cases there was a history of some injury or strain of the hip in adolescence which gave rise to trouble for a limited period, and then apparently recovered. In these cases the time of onset was reckoned from this early injury and not from the occurrence of stiffness and pain at a later age. It has been possible to find the old X-ray photographs (*Fig.* 494) or full notes of these injuries in only five cases; three of these show typical X-ray changes of osteo-chondritis

deformans juvenilis, or Perthes' disease, and in the other two there are distinct signs of a partial slipping of the neck of the femur on the epiphysis. In all five of these cases there has been an alteration in the normal joint relationship between the head of the femur and the acetabulum.

Normally the head of the femur is so shaped in relation to the socket that almost half of its articular surface is in contact with the acetabulum at any angle, but if this relationship has been altered by the widening and irregularity of the head of the femur, or by the sliding upwards of the neck, then more pressure will be borne on a restricted area, and alterations of the articular surfaces of the femur and acetabulum are the natural consequence. That pressure applied obliquely to a normal joint, or with an uneven distribution, may result in osteo-arthritis is well shown by the reaction of the ankle-joint to the strain cast on it by a malunited fracture of the leg, or, as instanced by Arbuthnot Lane, in the unilateral osteo-arthritis of the lumbar spine resulting from the constant carrying of heavy weights on one shoulder by dock labourers. There seems no doubt also as to the possibility of the occurrence of osteo-arthritis of the hip-joint resulting from one severe trauma, but strangely enough in very few instances does the patient associate his disability with one injury, however severe.

X-ray Appearances.—The X-ray appearances are usually typical; there is the loss of joint space due to the wearing away of the articular cartilages, atrophy of the bones due to a diminished lime content, and around the articular margins of the acetabulum and femur lies a rim of osteophytic outgrowths. In bilateral cases this is the X-ray finding with the head of the femur normal in shape and position, or approximately so, but in unilateral cases there is usually in addition some distinct alteration in the shape of the head of the femur. As a rule this is flattened and depressed with its convex surface forming a continuous curve with the upper surface of the neck of the bone, and the great trochanter is raised above its normal site as a result of some old-standing injury or deformity of the head or neck of the femur.

Etiology.—It seems clear then that in osteo-arthritis of the hip-joint we are dealing with two separate and distinct problems. In the toxic, or infective, arthritis there is the problem of a generalized infection which has led to widespread joint changes, and the causative infection—of whatever type—is probably still present. In osteo-arthritis of one hip—whether or not there has been any infective process—the exciting cause of the condition has probably been trauma, or an alteration of joint mechanics, so that the fear of spread of the condition to other parts is not present to the same extent. The two types—infective and traumatic—have many points of similarity; the X-ray changes show only minor differences in regard to bony outgrowths. Limitation of movements occurs equally in both conditions, but on account of their essentially different origins there are certain fundamental differences in the treatment which should be adopted for their relief.

It is with the object of clarifying our views as to the best line of treatment to be adopted for various types of osteo-arthritis of the hip-joint that the following review has been made of the treatment and after-results of 89 cases dealt with during the past fifteen years. This number includes unilateral and bilateral cases, in addition to a few in which the arthritis was associated with some type of spondylitis.

TREATMENT

In discussing treatment I do not propose to say much about general treatment, which is usually carried out in a routine manner in all these cases. Sepsis—from whatever source—must be sought for and if found must be dealt with efficiently. Physiotherapy by massage, radiant heat, ionization, mud baths, hydrotherapy, etc., may be tried, either singly or in rotation, but unfortunately they usually result in little or no success. Temporarily at least most of them appear to do good; after having the treatment the patient feels better and agrees that the joint is less tender and more free, but after an hour, or even less, the condition is as before until the next treatment is given. Sometimes if it is carried out in a spa where the patient is away from business worries, is on a diet, and is free from intestinal stasis, there may be a definite diminution in the discomfort felt in the joint, but if bone changes are present no form of non-surgical treatment can give permanent relief, or can produce any lasting improvement.

Our choice of possible surgical procedures may shortly be classified under the following six headings: (1) Manipulation of the joint in order to increase the range of movement; (2) Rest of the joint so as to reduce the inflammation present and diminish the pain; (3) The formation of a new joint at the site of the old one—arthroplasty; (4) The formation of a false joint close to, but not at the original site—pseudarthrosis; (5) Complete destruction of the joint and ankylosis by operative means—arthrodesis; (6) An alteration of the weight-bearing through the joint by means of an osteotomy—bifurcation operation. With such a wide selection of surgical procedures our difficulty usually is not in performing some definite surgical operation, but rather in choosing which of the many possible procedures is the most suitable in any particular case. The following résumé has been made in order that the choice may be something better than a matter of chance.

1. **Manipulation.**—When the condition is met with in its early stages the bone changes, as shown in the X-ray photographs, are usually slight. There may, indeed, be only a sharpening of the articular edges, and the patient complains not of a complete loss of movement but rather of an inability to perform some particular movement which he has previously been in the habit of doing regularly; there is often a difficulty in putting on or tying his shoe. Rotary movements, such as swinging at golf, may cause a sudden stabbing pain, and on clinical examination it may be found that only one movement of the joint is in any degree limited. In a case such as this (and in a patient otherwise healthy) gentle manipulation of the joint under an anæsthetic is likely to increase the range of movement and enable the patient to perform the acts which have previously been impossible. There is one very important clinical point to be taken into consideration before such a manipulation is undertaken. If the patient states that the pain caused by attempting the restricted movement is of short duration and disappears after a few minutes' rest, then the result of manipulation is usually satisfactory; but if he states that the pain only disappears after a considerable time, the result of such a manipulation will probably be very disappointing.

The manipulation must not be haphazard but must be carried out in a routine manner; the hip is gently moved through its whole range, each movement being performed once and once only. After-treatment consists in free use of the joint and exercises so designed that the movement which was limited previously is

performed at least three or four times a day. The result in a suitable case is often dramatic, and the patient is apt to praise unduly the manipulator, whether qualified or unqualified, and for a period he contrasts the great benefit he has received from the manipulation with the disappointing results of all previous treatment. The period of relief varies very considerably from a month to five or six years, but in the average case improvement lasts for about a year (*Figs.* 495, 496). If, however, manipulation is carried out in a more advanced case, no improvement is seen —in fact, the patient may only be made more tender and crippled than before.

This form of treatment was carried out in 27 cases of this series, with the following results: In 9 cases the result was a complete failure, as pain and limitation were increased and some other form of treatment had to be adopted

FIG. 495.—Early osteo-arthritis of the hip, relieved of pain by manipulation.

FIG. 496.—The same patient three years later on, showing that the condition of the hip has progressed. Further manipulation at the patient's own request, for a return of pain, was not successful.

within a few months. In 11 cases there was a definite improvement—pain was less and movements were increased for an average of one year after the manipulation. In the other 7 cases improvement continued for a period between three and five years, but in every case where it has been possible to find the subsequent history there has been a return of the disability with gradually increasing lipping of the joint as shown in the X-ray photograph, and in this stage a second manipulation would only increase the patient's discomfort.

2. **Fixation and Rest.**—When the condition is more advanced, with a more general limitation of movement and with definite X-ray changes, then manipulation of the joint—except when used to correct a deformity—cannot be of benefit, and some other form of treatment must be undertaken. Of all the possibilities open to us, by far the simplest is rest and support of the joint. This is commonly carried

out in one of two ways : Firstly—and in my opinion inadvisedly—by the application of a walking caliper which to some extent relieves the hip from body weight. This cannot prevent movements of the joint, and therefore has no effect on the inflammation which is the direct result of the rubbing together of two roughened and irregular articular surfaces. The second method is the application of a plaster spica in which the patient may walk. While this latter does not relieve any of the body weight, it is much more efficient than the former, because movements of the joint are prevented and the inflammation present is thereby reduced.

The latter treatment has been adopted in 24 cases ; in 20 of these the condition was a monarticular traumatic arthritis, and in the other 4 a painful infective arthritis. In the case of the 4 latter the result as far as relief from pain was concerned was

FIG. 497.—X-ray of a case of early osteo-arthritis of the hip, successfully relieved of pain by plaster spica for three months.

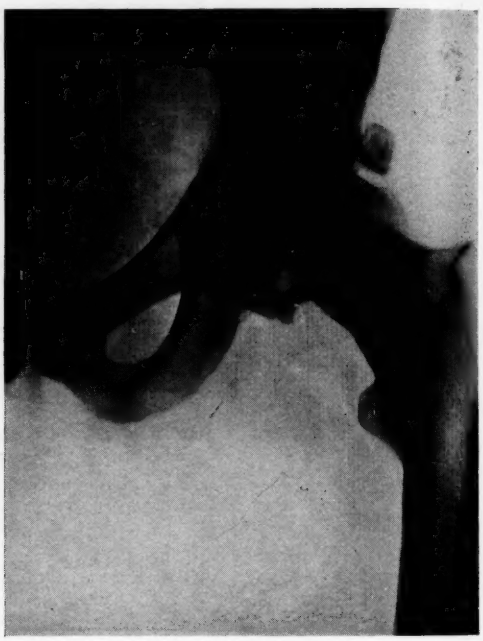


FIG. 498.—X-ray of the patient shown in *Fig.* 497 five years later, still free from pain, but the osteo-arthritis has progressed.

excellent, but while the pain was diminished or lost so also was movement, and ankylosis—complete or partial—was the invariable result. In the other 20 cases the result was good. In every case pain was temporarily diminished, and as a direct result of the rest of the inflamed joint the range of voluntary movement was increased in 6 cases. This increase of movement is of great interest as it illustrates the beneficial effects of the elimination of the constant irritation of the joint which is caused by the rubbing together of the roughened bony surfaces. In none of the cases of monarticular osteo-arthritis was there an appreciable permanent diminution of movement attributable to the prolonged fixation in plaster.

Unfortunately, although the treatment by fixation in plaster gave these excellent results it did not in any way alter the pathological conditions present in the joint (*Figs.* 497, 498), so that a recurrence of inflammation and pain took place

in most cases in a period of three months to eight years. The great advantage of this line of treatment is that there is no operation and no period of recumbency—two factors which are of importance in these patients, some of whom are unsuitable subjects for surgical operations, while others have a strong bias against operations of any type. During the wearing of the plaster spica exercise is permitted, and with the diminution of pain the patient can frequently walk about more freely than before the application, but there are undoubtedly certain disadvantages. Firstly, the application of a plaster spica which will remain in position on the hip of a very fat woman is usually extremely difficult, and secondly the weight of the plaster may be so great that the patient feels quite unable to carry around this extra load.

3. **Arthroplasty.**—Theoretically the ideal surgical procedure in every case of painful arthritis is the reshaping of the articular surfaces into their original contour, and the formation of a new joint, either with or without the interposition of some tissue such as fascia or fat, whereby the movements of the joint may approximate to normal. Many procedures have been described and much ingenuity has been shown in devising the correct technique required to obtain this ideal result, but unfortunately the average result of arthroplasty of the hip, however performed, has been decidedly poor.

In the series under discussion arthroplasty has been performed on 7 occasions during the past twelve years. In 5 cases the result was so indifferent that the patient eventually insisted upon some other treatment being adopted in order to improve the condition. In each case disappointment was expressed on account of the small range of movement possible in the joint, and also because even this range was not accomplished without discomfort. In 5 cases arthrodesis of the joint was necessary and was agreed to readily. The 2 remaining patients were satisfied, they were able to carry on their duties with comparative comfort, and although the result was not perfect, yet in each case the patient considered he was better than before the operation. The only movements possible were flexion and extension through a range of 30° and 40°; as the leg passed from the straight into the flexed position it also went into slight abduction and external rotation, but this was the only abduction or rotation possible to the joint. The X-ray photographs (*Figs.* 499, 500), which were taken five years and four years after the arthroplasty, show the great heaping up of new bone round the joint which is the cause of this loss of rotation. None of these cases of arthroplasty can be classified as anything but a failure.

4. **Pseudarthrosis.**—The operation of pseudarthrosis, or the formation of a false joint at some distance from the affected joint, is, however, in an entirely different category. It is an operation designed to give movement where movement is essential, as in a case of spondylitis in which one or both hip-joints are also affected, or when both hip-joints are involved and useful movement is not possible. It has been carried out on 4 occasions in this series, and in each case movement has been obtained, although usually at the expense of a certain amount of stability. This want of stability is the one great drawback to the wider use of this procedure. The operation is performed by removing a large portion of the neck and upper end of the shaft of the bone, and then implanting the great trochanter—to which the muscle group is still attached—on to the head and remaining portion of the neck. The upper divided end of the shaft of the femur now lies below the inturned outer surface of the great trochanter, which is completely covered by muscle attachments

and therefore cannot re-unite with the shaft of the bone. Such a procedure invariably gives movement, but owing to the loss of control of the gluteal muscles on the shaft of the bone there is an inevitable diminution in the voluntary power of the limb.

There are two definite indications for the operation: firstly, bilateral osteo-arthritis in which the legs have become so adducted that they cannot be separated, or may even actually cross each other; and secondly, when the lumbar spine also is involved in an extensive osteo-arthritis. When both joints are involved it will often be found that one hip is painless and firm while the other shows a small range of painful passive movement. If the operation is carried out in such a case, it should be performed on the painful side, leaving the painless limb as a sound

FIG. 499.—The result of an arthroplasty performed five years previously, showing the heaping up of bone on the rim of the acetabulum and consequent limitation of movements.

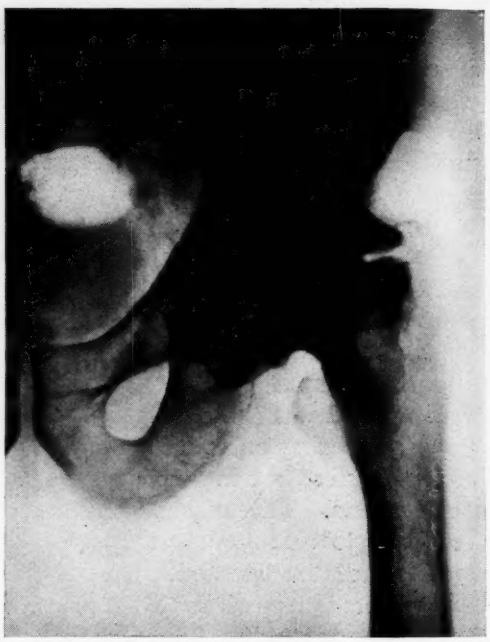


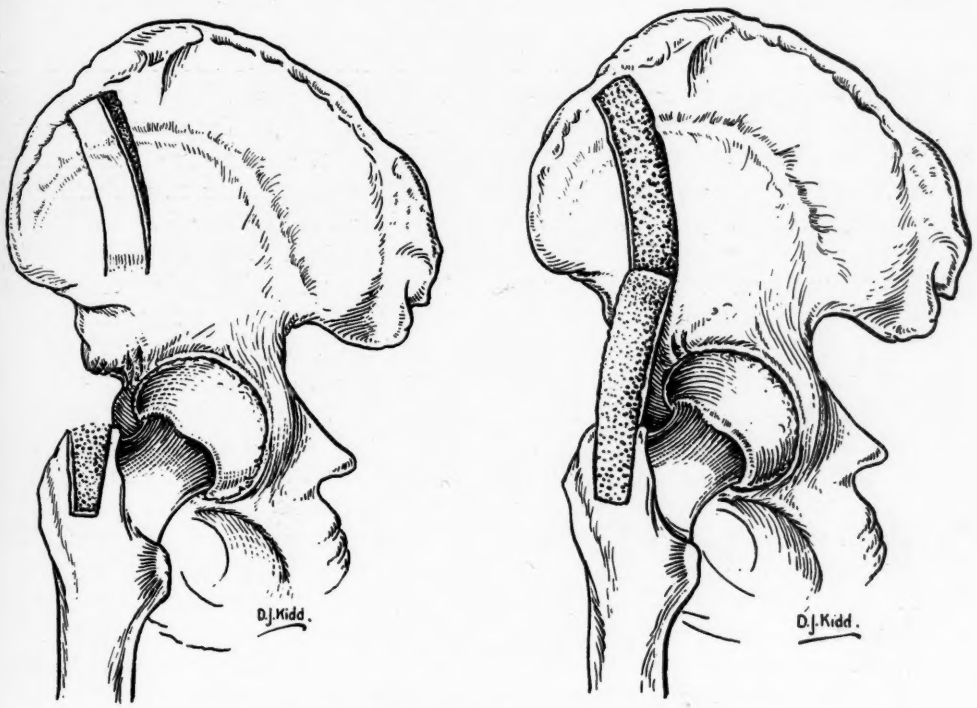
FIG. 500.—Similar result of an arthroplasty after four years.

weight-bearing pylon. Again, in some cases it is found that with bilateral affection one leg is definitely longer than the other, and here the indication is clear and the operation should be carried out on the longer side.

5. **Arthrodesis.**—When arthroplasty failed in the cases reported in this series we fell back instinctively on the operation of arthrodesis, and in all cases this gave a good result without pain, and with full stability. It might seem that this operation is the best for every case of painful monarticular osteo-arthritis of the hip-joint because it eliminates all pain from the joint, retains full stability of the limb, and leads to no shortening of the leg.

Including the 5 cases where an arthrodesis was performed following the failure of an arthroplasty it was performed on 17 cases in this series, and the patients were

highly satisfied with the result. It was not successful from the point of view of producing a bony ankylosis in 6 cases. These were all included in the group of 10 in whom an intra-articular arthrodesis alone was employed, so that of these 10, a bony union was secured in only 40 per cent, but in each of these 6 cases, although there was 5° to 10° of passive movement, the clinical result was excellent. Bony union was secured in each of the other 7 cases by means of the combined intra- and extra-articular arthrodesis, where, in addition to a complete removal of all the cartilage from the head of the femur and acetabulum and a widespread fragmentation of the cancellous bone underneath, a flap of ilium above the level of the hip-joint was turned down and attached to the femur in the region of



FIGS. 501, 502.—Extra- and intra-articular arthrodesis.

the great trochanter (*Figs.* 501, 502). Its base still formed part of the structure of the wall of the ilium and its blood-supply was preserved by this living continuity.

This method of combined operation makes for a more certain result (*Fig.* 503), and it is preferable to the single method where the head of the femur—which has been diminished in size—is placed in the much enlarged acetabulum, thereby giving a small area of bone-to-bone contact.

Although the operation of arthrodesis gives complete relief to the pain and deformity of the hip and the patient is highly pleased, it is not without its disadvantages. The following case illustrates this very well: The patient was suffering from a very extensive painful osteo-arthritis of the right hip, and in January, 1926, an arthrodesis was performed. Six months later she was able to walk freely and without pain, and this state of relief lasted for six years, until 1932, when she returned complaining of pain and aching in the lower lumbar spine, undoubtedly

due to the increased work thrown on this area by the absence of movements in the hip-joint. Unfortunately this is not an isolated case, as in 6 others there was a similar complaint of lower lumbar and sacro-iliac pain which was not present before the operation. The operation also is one of some magnitude, as it requires thirty-five to forty minutes at least for its accomplishment, and the lifting out of the head and cleaning out the acetabulum involve considerable shock.

6. **Bifurcation.**—The question of shock must be very seriously considered in those patients who are fat with weakened cardiac muscles, as such an extensive surgical procedure may be quite beyond their powers of resistance. A single case may be taken to illustrate the importance of this point. A lady 52 years of age

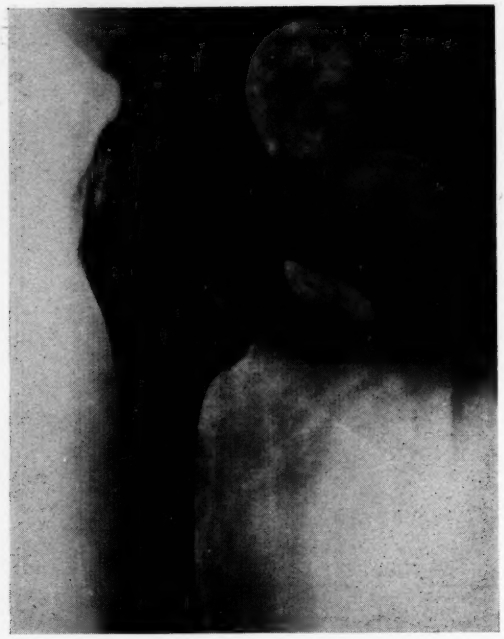


FIG. 503.—Result of an intra- and extra-articular arthrodesis of hip.

and 13½ stones in weight, had been under the care of a physician for the previous four years on account of slight myocardial degeneration. She developed an extremely severe condition of osteo-arthritis of one hip so that she was unable to walk more than a few steps in her bedroom. As she was so exceedingly stout it was impossible to fit her with any form of hip shield or plaster spica, and it was decided that if at all possible some form of surgical treatment was advisable. After consultation it was decided that for her the only safe anæsthesia was gas and oxygen, which she might stand for not more than fifteen to twenty minutes. The operation of Lorenz bifurcation was successfully employed on this occasion, and she is now able to walk without pain for a quarter of a mile, and her general health is much improved as a result of the exercise which the operation has rendered possible. It is now largely owing to this consideration of absence of shock that I perform the operation of Lorenz bifurcation in certain suitable cases of mon-articular osteo-arthritis, and this procedure has been carried out on 15 occasions.

The operation has many great advantages; it can be performed in a few minutes; the shock is negligible; and the recumbency after the operation need not be so long or so continuous as it must be after arthrodesis of the joint.

Technique.—An incision 6 to 7 in. in length is made from the tip of the great trochanter along the line of the femur, and the muscles in this area are partially stripped by blunt dissection so that the level of the neck and head of the femur may be ascertained. An oblique osteotomy is now made in the shaft of the femur, passing from below upwards and inwards at such a level that the inner end of the lower fragment lies about ½ in. below the level of the head of the femur (*Fig.* 504). The upper end of the lower fragment is now displaced inwards and slightly upwards by means of a smooth lever and by abduction of the leg so that the shaft of the femur comes to lie just below the lower border of the acetabulum (*Fig.* 505), and unless this position of the shaft is obtained the result will be unsatisfactory. In this situation the shaft of the femur lies outside the capsule of the joint and there is no fear of union occurring between the shaft and the acetabulum. Originally this operation was performed as a subcutaneous osteotomy and the final position of the lower portion of the bone was left largely to chance. This is the most common source of failure, and the operation should always be carried out through an incision sufficiently long for a careful inspection of the whole site of operation, and for the verification of the correctness of the final site of the femoral shaft.

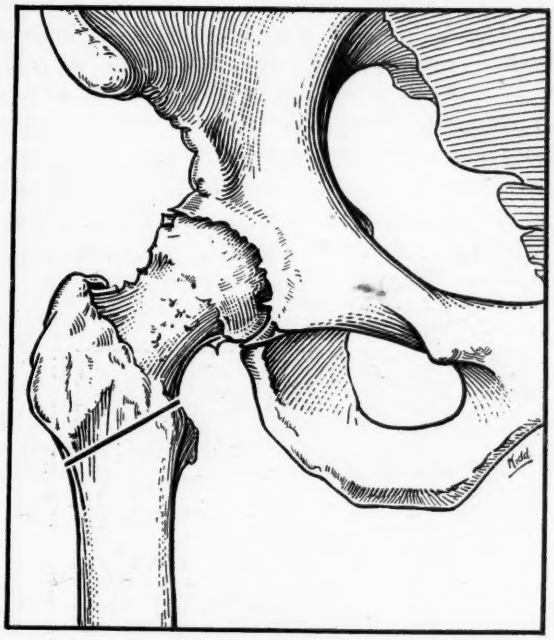


FIG. 504.—Diagram showing site of osteotomy for Lorenz operation.

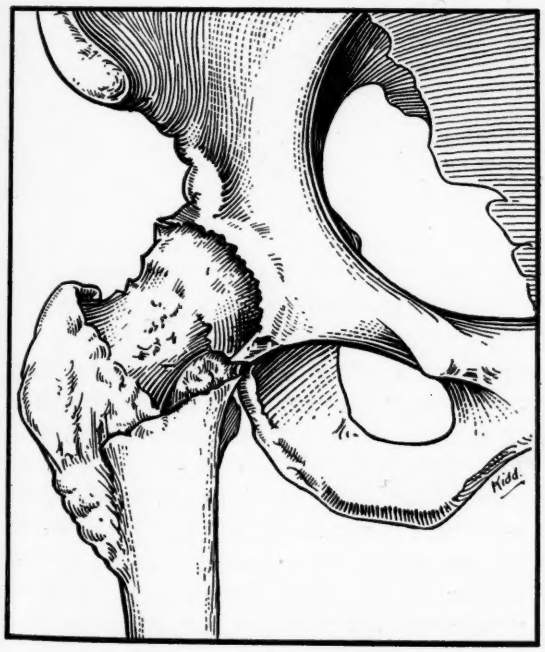


FIG. 505.—Diagram showing position of the fragments after a successful Lorenz osteotomy.

There are certain essential

conditions for success in this operation: (1) The new site of the upper end of the shaft of the femur must be just below the edge of the acetabulum; (2) There must be union between the portions of the divided femur, as otherwise a weak painful hip-joint is the invariable outcome. After the division of the bone the lower fragment is pushed inwards into its new position; no effort is made to change the position of the upper fragment, which tends to follow after the lower fragment as the two fragments have so many muscular attachments in common. The leg is then fixed in plaster with the hip neither abducted nor adducted, and after a suitable rest of four to five months in plaster—during the latter two months of which time the patient is allowed to get about with weight-bearing—the plaster is removed and complete freedom is allowed. The two

FIG. 506.—X-ray of a case of osteo-arthritis of hip before a Lorenz osteotomy was performed.

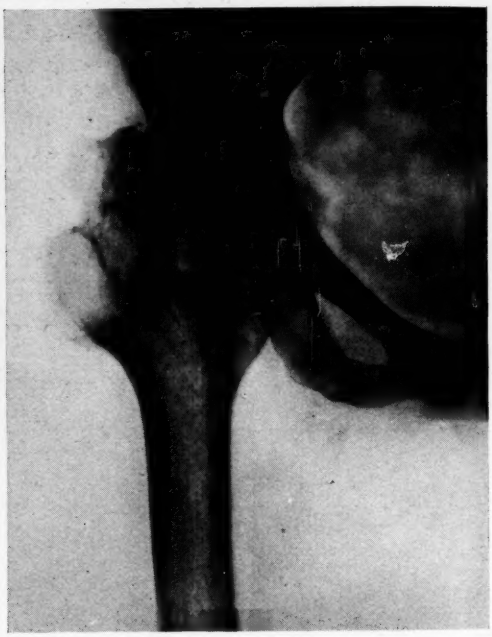


FIG. 507.—X-ray of case shown in *Fig.* 506 two years after a Lorenz osteotomy was performed. The patient is entirely free from pain and has been at work in a shop for the last year.

portions of the femur have now joined; movement which may have been present in the joint is not lost but is taking place through a different angle, and the relief is almost as great as after the operation of arthrodesis, without many of the very great disadvantages of this operation (*Figs.* 506, 507).

Results.—In 12 cases the result has been excellent; the pain has gone, movement although reduced is still present, and there is not the same tendency to strain the lumbar and sacro-iliac regions. In my opinion the operation holds a very important place in the treatment of osteo-arthritis of the hip-joint. In 3 cases the results have not been satisfactory; pain was not removed and deformity was still present (*Fig.* 508). The reason in each case was the same—namely, the operation incision was too small, so that the whole area could not be inspected and the

shaft was not placed in the correct position. As a result body weight continued to pass through the hip-joint as before. This error can be prevented by a larger incision, by inspecting the area of operation, and making sure of the final situation of the upper end of the lower fragment of the femur. If this is done, the result will almost certainly be satisfactory.

FIG. 508.—X-ray of an unsuccessful result following a Lorenz osteotomy, showing the shaft not displaced far enough under the acetabulum and failure of correction of the line of weight-bearing.

CONCLUSIONS

Manipulation may produce a relief of symptoms and increased movement of a temporary nature in cases of osteo-arthritis.

Arthroplasty is the ideal operation, but at present our methods and technique fail to secure any real benefit for the patient, and the operation is usually a failure.

Arthrodesis removes the pain and deformity and greatly increases the stability of the joint, but it is a very extensive procedure, and, if successful, results in a great increase of strain on the lumbar spine and sacro-iliac region.

The bifurcation operation of Lorenz is simple, of short duration, and if performed correctly leads to a relief of pain and deformity with no loss of stability, and does not cause strain on the lumbar region.

THE PATHOGENESIS OF PAGET'S DISEASE OF THE NIPPLE AND ASSOCIATED LESIONS

By Sir ROBERT MUIR,
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During the last few years I have been engaged in a study of Paget's disease and other affections of the breast and have published several papers (1927–34) dealing especially with the former condition. It may, however, be well to give a short general account of my views and the observations on which they have been based, especially in relation to the surgical aspect, and in doing this I have added some new observations along with some new illustrations. I have not made an attempt to deal with the extensive literature on the subject.

In my first paper (1927) reasons were given for coming to the conclusion that in the great majority of cases Paget's disease results from an extension of cancerous proliferation from the ducts of the nipple ('intra-duct carcinoma') to the surface epidermis of the latter—an overflow of cancer cells, as it were, to the epidermis with subsequent spread and multiplication therein. I have said "in the great majority of cases" because I admitted the possibility of the occasional occurrence of a primary epidermal carcinoma with intra-epidermal spread such as is met with on other parts of the body. This, however, is referred to below.

To the surgeon the most important practical point is the tendency for Paget's disease to be associated with or followed by carcinoma in the substance of the breast, and the relation of these two occurrences has often been the subject of discussion. In connection with this it should be observed that in his original paper Paget not only gave an accurate account of the naked-eye appearances of the nipple lesion but noted that carcinoma frequently ensued and that the site of the malignant growth had no relation to the nipple, but might be deep in the substance of the gland. (As a matter of fact, carcinoma developed in all his cases.) What, then, is the relation between the Paget lesion and the carcinoma? If the former is a primary lesion of the epidermis, I can see no possibility of any causal relationship. If, on the other hand, they are both secondary to the same condition—namely, intra-duct carcinoma—their association receives at once a ready and an intelligible explanation. Accordingly, if we use the term 'Paget's disease' in a sense more in conformity with his original account—a characteristic nipple lesion *liable to be associated with mammary carcinoma*—I believe that the association of the two conditions, Paget's disease and carcinoma of the breast, is due to both being sequels to antecedent malignant proliferation within the ducts of the gland. When the Paget lesion is present the surgeon should have in his mind the picture of this proliferation within the ducts—a proliferation which may break through the walls at any time and give rise to an ordinary infiltrating carcinoma, if in fact this has not already occurred.

As the Paget cells in the epidermis are the characteristic feature of the disease I may anticipate what follows by stating at this point that similar cells are met with within the epithelium both of the ducts and of the acini. In all these situations their appearance and behaviour are essentially of the same nature. The only important difference is that the Paget cells in the epidermis undergo degenerative changes as they pass or are carried towards the surface and thus become the well-known small and sharply contoured structures. In the epithelium of the ducts and acini, on the other hand, they remain in a more healthy and active state and resemble the active Paget cells in the deepest layers of the epidermis. We may thus give a general definition of a Paget cell as *a cancer cell growing within a healthy or at least non-neoplastic epithelium.*

As in my view intra-duct carcinoma is a condition antecedent both to Paget's disease and ordinary carcinoma, it will be convenient to consider first its main features—its characters, modes of spread, and secondary results.

OCCURRENCE AND CHARACTERS OF INTRA-DUCT CARCINOMA

I have applied the term 'intra-duct carcinoma' as meaning a malignant proliferation of duct epithelium before it has broken through the normal limits, i.e., the walls of the ducts, and infiltrated the tissue spaces. The term seems to me a justifiable one and the most convenient. The use of the term 'carcinoma' may appear misleading, but it should not be so if the qualifying adjective is kept in view. As I have said elsewhere, epithelial cells do not become malignant on getting into the tissue spaces; they invade the latter because they have acquired the essential characters of malignant neoplasia, and they acquire this character before they transgress the normal boundaries. It is true that the sure sign of malignancy is infiltration, but when one keeps in view the two facts—namely, that the malignant property is developed before actual infiltration occurs, and, secondly, that the histological features of the cells in question correspond whether they are within the ducts or outside in the tissue spaces—then the term 'intra-duct carcinoma' is both justifiable and of descriptive significance. It is preferable to the term 'duct carcinoma', as the latter may signify merely an ordinary carcinoma which has arisen from the epithelium of the ducts.

Intra-duct carcinoma may be regarded as the final stage of epithelial hyperplasia often met with in chronic breast disease and often associated with a varying degree of fibrosis and cystic change. The stages of such epithelial hyperplasia have been described in detail by Cheatle (1925–31) and by Charteris (1930), and I agree in all essential points with their accounts. It is sufficient to say that all transitions from simple papillomatous ingrowths in the ducts to intra-duct carcinoma are found. We meet with papillomata with broad connective-tissue base and also those with filamentous supporting connective tissue, but in both types there is always co-ordinated growth of epithelium and connective tissue and the normal relationship of these is maintained. Further stages are those in which the stroma gradually diminishes in amount until one meets with epithelial proliferation without any connective tissue between the cells. The cells may form definite projections from the wall of the duct, or again they may form masses in which polarity of the

cells is still shown by the formation of acinus-like circles, or, lastly, the cells may have no definite arrangement. The cells are at first fairly uniform in size and in their nuclear characters, but later show great variations both in size and in form. The nuclei also come to vary; many are enlarged, pale, and of vesicular type, whilst aberrant forms also are met with. Ultimately the duct may be filled with a mass of cells showing all the histological characters of encephaloid carcinoma (*Figs.* 509, 510). It is to this stage of epithelial hyperplasia that I consider the term 'intra-duct carcinoma' may properly be applied. It is quite evident that such cells may break through the walls of the containing duct and infiltrate the tissues, thus giving rise to ordinary carcinoma of the breast; and, as a matter of fact, the actual break through may occasionally be seen.

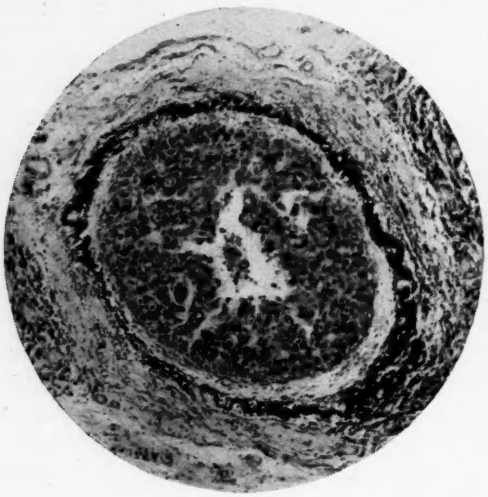

FIG. 509. (× 75.)

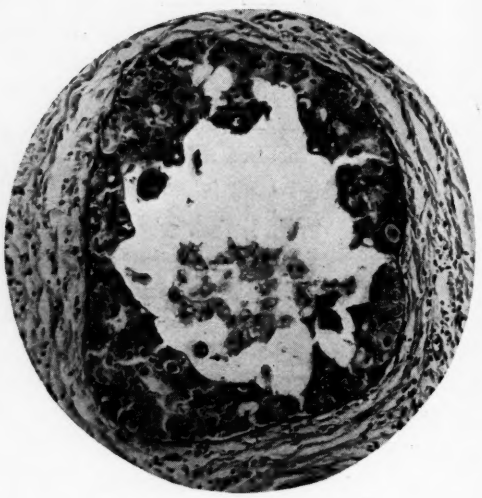

FIG. 510. (× 90.)

FIGS. 509, 510.—Transverse section of two intramammary ducts, the seat of intra-duct carcinoma, showing characteristic appearances. In *Fig.* 509 there is some hyperplasia of the elastica.

It may also be mentioned that the cells undergoing malignant proliferation may extend from the small ducts into the acini and may grow within the latter. In this way there may occur what is practically a replacement of the epithelium of the acini by cancerous growth, the normal structural outlines of the acini being still maintained. We may then speak of an *intra-acinous carcinoma*. I have seen such a condition on an extensive scale without there being any actual infiltration of the connective tissues.

I may refer to one point in connection with the interpretation of the changes described. In cases where ordinary carcinoma is present and the ducts also are filled with cancer cells, the view has been taken by some writers that the ducts have been invaded by cancer cells. The possibility of such an occurrence as an occasional event cannot, of course, be denied, but I have no doubt that it is exceptional, and that the sequence of events is of the opposite kind—the cancer outside is secondary to the disease within the ducts. The following reasons for this view may be given. In a case where ordinary carcinoma is present the various stages of evolution of intra-duct carcinoma may sometimes be followed; epithelial hyperplasia terminating

in malignancy may be seen. We can scarcely suppose that cancer cells after penetrating ducts from outside lose their anaplastic characters and become more differentiated again. But more conclusive evidence is that intra-duct carcinoma may be present, and often is, without any infiltrating carcinoma of the tissues being discoverable. I have seen the combination of extensive intra-duct carcinoma in the breast with spread to the acini, intra-duct carcinoma of the nipple and Paget's disease, without any infiltration of the connective tissue. Or again, and this is a stage more frequently met with, ordinary carcinoma may be associated with intra-duct carcinoma in one area while intra-duct carcinoma alone is present in other parts of the breast, and, further, may show different stages of evolution. I consider, therefore, that in the vast majority of cases where intra-duct carcinoma and ordinary carcinoma are present, the latter is secondary to the former.

There is no doubt that intra-duct carcinoma is a condition of frequent occurrence. I have found it in the large majority of cases of ordinary breast carcinoma which I have recently examined, and, in view of the fact that a small area showing the lesion may escape detection, I have formed the opinion that it is usually present. Its distribution varies greatly. It may occur in a localized area, often attended with fibrous induration and cyst formation, or several such areas may be present, or again it may affect a considerable part of the breast. When it is present in some ducts, epithelial hyperplasia at various stages of evolution may be present in others. It may affect the ducts of the nipple and those in the underlying tissue as well as in the breast itself, and occasionally the former may be affected alone. It will be clear from what I have stated that it is only when the upper parts of the ducts or of a duct in the nipple are affected that Paget's disease occurs as a secondary phenomenon. It may be definitely stated also that intra-duct carcinoma, the final stage of epithelial hyperplasia, occurs at an earlier period of life than does ordinary carcinoma. This has been insisted on by Cheatle, and I agree fully with him. As an illustration, in the five cases of Paget's disease on which I based my original views and in all of which duct carcinoma was present, the age of the eldest patient was 48 years.

It is clear, too, that intra-duct carcinoma is a very chronic condition and may exist for many years without breaking through the walls of the ducts; it may, in fact, never break through at all. Further, in a recent paper (1934) I have shown that in some cases the growth may at places undergo retrogression and disappear, this process being accompanied by overgrowth of the connective tissue internal to the elastica of the duct wall, complete obliteration sometimes following. If my views with regard to Paget's disease are correct, then in cases where this disease is present we get another indication of the chronicity of the intra-duct carcinoma which precedes it for a considerable time. For example, I have recently met with a case in which the Paget lesion was present for ten years. No ordinary carcinoma could be found after careful microscopic examination, but extensive intra-duct carcinoma was present, and in some of the ducts affected by it obliterative changes had occurred.

To sum up, then, intra-duct carcinoma is a common affection of chronic nature, its time of incidence preceding the cancer age by a considerable period. It often breaks through the normal confines of the ducts and gives rise to ordinary carcinoma, but may exist for a long period without doing so; occasionally it leads to the relatively uncommon condition of Paget's disease.

MODES OF SPREAD OF INTRA-DUCT CARCINOMA

This is a matter of much importance in relation to Paget's disease. I have already stated that malignant proliferation within ducts varies much in its distribution. It may occur in different parts of the breast, may affect considerable areas, may occur in the ducts of the nipple or of the breast or of both together. We have thus to consider how this varied distribution is brought about. There appear to me to be three main factors concerned :—

1. In the first place, the malignant change may arise in multiple foci ; of this there can be no doubt. Thus it is met with in different parts of the breast which are anatomically distinct and is also met with in several ducts in the nipple and beneath it, occasionally in a considerable proportion of these ducts. We know nothing of the essential cause or influence which acts on the epithelium and gives rise to the malignant change, but, whatever be its nature, it may clearly act on different portions of the breast at the same time.

2. In the second place, when the epithelial cells have assumed the neoplastic character they may grow in masses within the ducts and come to form a sort of injection of them ; occasionally the same is met with in the acini.

3. But, in the third place, there is another mode of spread, namely, a spread of cells with malignant properties in relatively healthy epithelium—*the intra-epithelial spread of carcinoma.* This is an occurrence which is not in any way peculiar to the disease under consideration, but is seen in other conditions where the true nature of the phenomenon does not admit of any doubt. This process of intra-epithelial growth is in my opinion all-important in connection with the changes which we are considering, and some facts with regard to it may now be given.

By intra-epithelial growth, then, we mean the invasion of normal epithelium by cancer cells ; the latter grow within the epithelium and destroy it to a varying extent, the epithelium playing a passive rôle. The cancer cells may occur in masses in the invaded epithelium or they may be disseminated and occur singly, this latter condition being apparently due to their wandering in the epithelium. Such a mode of growth is now coming to be generally recognized. I have elsewhere written on the subject (1930), but the main facts with regard to its occurrence may be summarized as follows :—

1. In the first place, carcinoma may start in an epithelium and grow within it. This is best illustrated by primary intra-epidermal carcinoma about which much has been written by dermatologists in recent years. Bowen's disease is one well-recognized example, but there are other varieties. The cells of the growth are distinguishable from the normal epithelium within which they grow in masses or in a scattered form ; they present different morphological features and there may be aberrations in type. The growth is of a very chronic character, often going on for years ; occasionally infiltration of the cutis vera follows, an epithelioma, basal-cell carcinoma, or a growth of other type resulting. Primary intra-epidermal carcinoma, though uncommon, can no longer be regarded as a rarity.

2. Secondly, a malignant growth infiltrating the cutis may invade the epidermis and spread within it ; this is a rare occurrence, but when it is met with the appearances are of striking nature. Carcinoma of the breast may occasionally invade the epidermis in this way, and the invading cells then behave in a fashion analogous

to what is seen in Paget's disease. The most striking example I have seen of such a secondary epidermal invasion is that described by Shaw Dunn (1930) in the case of a mucoid (colloid) carcinoma of the rectum. In the drawing accompanying his paper the cancer cells, stained red with mucicarmine, are clearly picked out from the epidermal cells and their distribution is seen at a glance. No clearer or more convincing demonstration of what I must call the wandering of the cancer cells could be had. A similar phenomenon is occasionally seen in malignant melanoma, as has been described by others. I have recently seen such a case, the growth being at the anal orifice, and the pigment-forming malignant cells are distributed in the epidermis just as in Shaw Dunn's case.

3. In the third place, the epidermis of the nipple may be invaded by cells of an intra-duct carcinoma in the upper part of a duct of the nipple—the route of spread in this case is an intra-epithelial one throughout. This I hold to be the ordinary mode of origin of Paget's disease, and it may now be considered more in detail.

INTRA-EPITHELIAL SPREAD OF CARCINOMA OF THE BREAST AND ITS RESULTS

In giving an account of this subject, it seems a good plan to start with the changes in the ducts in the breast and trace them to the surface of the nipple. It has already been stated that intra-duct carcinoma is a common primary event in malignant disease and that in its spread intra-epithelial growth may be concerned. This is shown by the presence of cancer cells growing in healthy epithelium where intra-duct carcinoma is spreading. The cells are often large and of rounded or oval form with vesicular nuclei and they occur in small collections and singly. In many cases they are sharply marked off from the invaded cells which become pressed upon and atrophied; one can often see the nuclei of the latter flattened and degenerating between the cancer cells. At a later stage of growth a duct may be lined by a sheet of cancer cells several layers in depth, whilst the fact that the growth is really intra-epithelial is shown by a surviving layer of the original epithelium on the luminal surface. Intra-epithelial growth may also be found in the acini of the breast, and here the large pale cancer cells can be clearly seen within the otherwise unchanged epithelium (*Fig.* 511). Occasionally, collections of acini are replaced by groups of cancer cells still within normal confines; they are usually surrounded by an abundant collection of lymphocytes and plasma cells. A study, then, of the breast alone, quite apart from Paget's disease, shows that the epithelium, both of the ducts and of the acini, may be invaded by cancer cells which have not

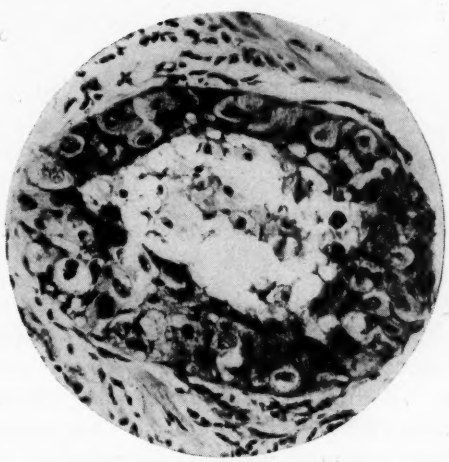

FIG. 511.—Mammary acinus, the seat of intra-epithelial growth of cancer cells. The cancer cells ('Paget cells') with clear protoplasm are seen growing in the epithelium of the acinus. (× 200.)

yet grown out into the tissue spaces. Such a mode of growth corresponds with that in the epidermis of the nipple now to be described and is in conformity with the definition of a Paget cell given above. Other illustrations of intra-epithelial growths in ducts and acini will be found in my first paper and in a paper by Simard (1930).

Spread to the Epidermis.—If intra-duct carcinoma spreads thus within the epithelium of ducts and acini, an important question comes to be: What will happen when the growth reaches the orifice of a duct? Let us keep before us a picture of the histological features at this position. The columnar epithelium at the orifice of a duct is, of course, continuous with the deeper cells of the epidermis and there is a transition between the two, sometimes relatively abrupt, sometimes more gradual. The transition, too, occurs at varying levels, the stratified squamous epithelium sometimes extending for some distance down the duct. When the growing cancer cells reach this junction, as often occurs, there are three possibilities: (1) The growth of cancer cells comes to an end; (2) The cancer cells may break through into the cutis and give rise to an infiltrating carcinoma; (3) The intra-epithelial growth may continue. With regard to the first possibility, one can say nothing. One may sometimes trace an intra-duct carcinoma only up to the orifice of the duct, but one cannot say that the growth has stopped. Again, the possibility of ordinary carcinoma arising at this site cannot be denied, but I have never seen any trace of this. The third possibility is the ordinary occurrence. The intra-epithelial growth of cancer cells goes on, and of necessity the cancer cells pass from the duct epithelium into the epidermis. Thereafter they spread and produce all the features of Paget's disease.

FIG. 512.—High-power view of deep portion of epidermis of nipple in Paget's disease. Note the large Paget cells of active appearance invading the rete Malpighii and sharply defined from the cells of the latter. (× 600.)

The relation of the cancer cells invading the epidermis to the resulting effects needs only a short account. The cells are in a resisting tissue, a relatively unfavourable environment, and they spread slowly. They can flourish only in the more cellular layers and especially in the deep part of the stratum Malpighii (*Fig.* 512). As the cells of the epidermis grow and move towards the surface the Paget cells are carried with them; the latter degenerate as they move upwards and come to form the sharply contoured structures so well known. The Paget cells spread widely in the epidermis over the nipple and grow both in clumps and as widely scattered solitary cells. The latter appearances, as I have indicated above, can be explained only on the view that they actively wander. Sometimes the cells are large and show active division, mitoses being present. They often correspond closely in appearance with the cancer cells in the upper extremities of the ducts. Sometimes,

however, nearly all the Paget cells are in a more or less degenerated condition, and then from such a case alone it would not be possible to interpret the appearances. It is only by studying a series of cases that once can trace the various modifications which the cells undergo.

As a rule the Paget cells are oval or rounded, in fact quite anaplastic, but occasionally one sees a return to a columnar type. It is not uncommon to find traces of this in the deeper layers of the epidermis, where the invading cells may form an imperfect layer resting on the cutis. Evidence of this is shown in *Fig.* 513, but *Fig.* 514 is a more striking example. The columnar epithelium in the latter encloses a small space containing fluid, the epidermal cells being raised above it. We have really an example of a small metastasis of adenocarcinoma, the cells of

FIG. 513.—Paget's disease of nipple. In the deeper part of the epidermis the Paget cells show transitions to the columnar type. (× 200.)

FIG. 514.—Paget's disease of nipple. The Paget cells form a distinct layer of columnar type resting on the connective tissue, with a space containing fluid above them. (× 200.)

which have reached the epidermis by the intra-epithelial route. In other parts of the epidermis all transitions to Paget cells of ordinary type were present. Such appearances seem to show clearly that the malignant cells are not of epidermal origin and confirm the view which I have put forward.

As soon as the malignant cells are present in any part of the epidermis a cellular reaction occurs in the connective tissue below. There is marked accumulation of lymphocytes and plasma cells; we find also new formation of blood-vessels and increased vascularity. The invading cells in some way irritate the tissue and a certain amount of exudation on to the surface of the epidermis occurs. These changes result in the red and raw-looking surface and the serous discharge characteristic of the disease.

Since my first paper was published I have examined about twenty cases of Paget's disease of the nipple, and observations on these have merely confirmed the view originally expressed. In all of them intra-duct carcinoma was present in the nipple, and in a large proportion direct spread of the carcinoma from the ducts to the epidermis could be traced. As a striking illustration of this, reference may be made to what was found in one case. In *Fig.* 515 there is shown a duct of the nipple cut longitudinally in its whole length with the exception of a small portion at its upper end; the depression in the epidermis indicates the position of the orifice. The duct throughout the portion shown is filled with cancer cells, and in the deepest part of the epidermis at the depression there are several small collections of large pale cells. A high-power view of these cells is shown in *Fig.* 516. As is clearly seen, they are large and appear pale and present a sharp contrast

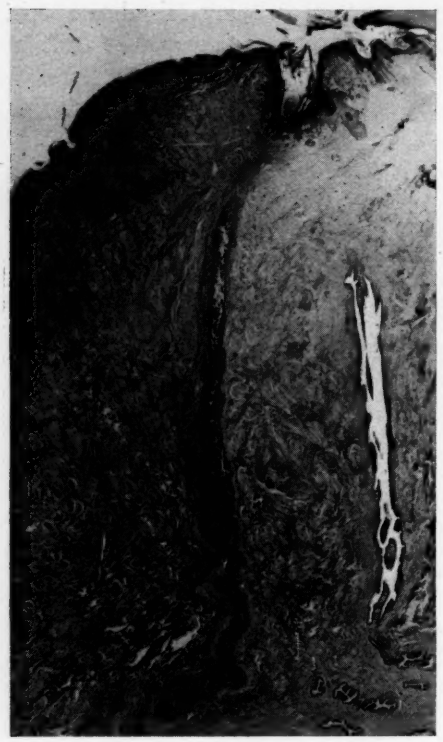

FIG. 515.—Longitudinal section of two ducts in nipple. The one on the left is the seat of intra-duct carcinoma and is almost filled with cancer cells. A few cancer cells in small groups—Paget cells—are present in deep part of epidermis at duct orifice (cf. *Fig.* 516). (× 10.)

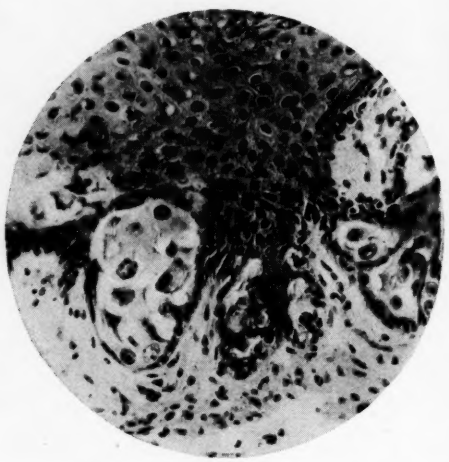

FIG. 516.—High-power view of deep portion of epidermis at orifice of duct shown in *Fig.* 515. Note the collections of Paget cells with clear protoplasm sharply marked off from the epidermal cells. The Paget cells were present only in this situation. (× 200.)

to the cells of the epidermis. They were found to correspond in all histological details with the cancer cells in the duct. We have here a picture of the earliest implication of the epidermis, in fact the cancer cells have just arrived in its deepest part. It does not appear to me possible to interpret the appearances shown in these figures in any other way. It may be added that there had been in this case no suspicion of the presence of a Paget lesion, and further that no Paget cells could be found at any part of the epidermis except at the orifice of the duct.

As stated at the outset, I do not deny the possibility of the occurrence of primary intra-epidermal carcinoma of the nipple such as is sometimes met with on other parts of the skin surface. I have met with only one case of this kind and

it was in a male subject. The underlying glandular tissue showed no lesion, and the significant fact is that a rodent ulcer had developed in connection with the skin lesion, an occurrence which is recognized in connection with such growths elsewhere. On the other hand, I have never seen basal-cell carcinoma or epithelioma or any form of invasive epidermal growth arising in connection with Paget's disease of the female breast.

SUMMARY

From the above account it will be seen that in the evolution of Paget's disease there are two main factors concerned, namely, *intra-duct carcinoma* and the *intra-epithelial mode of spread of this growth*. The main features of intra-duct carcinoma, a lesion of frequent occurrence, and its varying distribution have been described. The intra-epithelial mode of spread occurs in the epithelium of the ducts and acini as well as in the epidermis of the nipple—the ' Paget cells ' are cancer cells growing in non-neoplastic epithelium. Paget's disease occurs only when the intra-duct carcinoma is present in the upper portions of the ducts in the nipple, and is due to the spread of the cancer cells from ducts to the epidermis by the intra-epithelial route. Intra-duct carcinoma in this situation is relatively uncommon, and hence Paget's disease is somewhat rare. Much more frequent is the occurrence of intra-duct carcinoma in the ducts within the mamma, and then the ordinary result is a direct break-through of the cells with the production of an ordinary infiltrating carcinoma. Intra-duct carcinoma may lead to one or both lesions, and thus Paget's disease may precede or may follow ordinary mammary carcinoma, or again may develop without the occurrence of the latter.

BIBLIOGRAPHY

CHATERIS, A. A., *Jour. Pathol. and Bacteriol.*, 1930, xxxiii, 101.
CHEATLE, G. LENTHAL, *Brit. Jour. Surg.*, 1925–6, xiii, 509.
CHEATLE, G. LENTHAL, and CUTLER, M., *Tumours of the Breast*, 1931. London.
DUNN, J. SHAW, *Jour. Pathol. and Bacteriol.*, 1930, xxxiii, 1297.
MUIR, R., *Ibid.*, 1927, xxx, 451.
Muir, R., *Ibid.*, 1931, xxxiv, 594.
MUIR, R., *Brit. Med. Jour.*, 1930, ii, 587.
MUIR, R. and AITKENHEAD, ANNE C., *Jour. Pathol. and Bacteriol.*, 1934, xxxviii, 117.
SIMARD, CH., *Bull. Assoc. franç. pour l'Etude du Cancer*, 1930, xix, 50.

PARAPLEGIA IN POTT'S DISEASE, WITH SPECIAL REFERENCE TO THE PATHOLOGY AND ETIOLOGY*

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PARAPLEGIA is the commonest and most distressing of the graver complications of tuberculous disease of the spine. To the patient the onset of 'paraplegia' seems to herald his worst forebodings. Percivall Pott,[26] writing in 1779, summed up the situation from the layman's point of view in a single sentence which needs no amplification to-day: "If the patient be an infant, it becomes an object of constant though unavailing distress to its parents; if an adult he is rendered helpless to himself, and useless to all others, which of all possible states is surely the worst."

SOURCES OF CLINICAL MATERIAL AND STATISTICAL SURVEY

Gross Incidence of Paraplegia in Pott's Disease.—The gross incidence of paraplegia has been estimated to be as high as 20 per cent by some and as low as 5 per cent by others. Such observations will vary with the ages and the stage of the disease of the patients forming the bulk of any given series, and with the conditions under which they are seen, whether for a short time only or over long periods. For statistical purposes a survey has been made by the writer of 800 patients with Pott's disease from four different hospitals, forming at each a continuous series.

St. Thomas's Hospital (in-patients)	(1922–32)	182
Pyrford Orthopædic Hospital	(1922–32)	82
Queen Mary's Hospital, Carshalton	(1927–32)	206
Shropshire Orthopædic Hospital, Oswestry ..	(1927–32)	331
Total number with Pott's disease		801

Among these 801 were 92 with paraplegia, giving a gross incidence of 11·4 per cent. For further analysis there have been added to this series of 92 patients a further 94 collected by H. J. Seddon from other hospitals, making a combined series of 186 patients with paraplegia. Seddon's 94 patients were collected from the Royal National Orthopædic Hospital and the country branch of this hospital at Stanmore; St. Luke's Hospital, Lowestoft; Princess Mary's Hospital and the Royal Sea Bathing Hospital, Margate; and St. Vincent's Orthopædic Hospital, Pinner. Among these 186 patients were 7 that had a second paralysis at some

*The theses from which this and the following article are taken were both awarded the Robert Jones Medal and Prize last year by the British Orthopædic Association. The authors have collaborated for the purpose of the present publication, having eliminated the duplication of those parts which were common to both theses.

time after recovery from the first, so that the combined series contains 193 paraplegias in 186 patients. Patients with paraplegia were also studied at the Royal Cripples Hospital and the Woodlands Hospital, Birmingham, and Addenbrooke's Hospital, Cambridge, but these fell into no continuous series. Material for pathological study was obtained from many of the above-mentioned hospitals and from the museums of pathology of the London Medical Schools.

Incidence of Paraplegia in Relation to Age.—Tuberculosis of the spine has its onset much more commonly in childhood than in adult life. Onset is more frequent in the third than in any other year, and after the sixth year the incidence declines rapidly. According to Sorrel[31] 64 per cent of all Pott's disease starts in childhood. As the onset of disease is commoner in childhood than in later life, it might be expected that paraplegia would also be more common in children. The paraplegia may, however, supervene many years after the onset of disease, and actually it occurs in this series of 188 patients about equally under and over the age of 16.

Incidence of Paraplegia in Relation to the Level of the Disease in the Spine.—Paraplegia is rare with disease below the level of the 1st lumbar vertebra, since the cord has terminated below this point. Thoracic disease is far more common than cervical, and therefore the majority of paraplegias accompany thoracic lesions. In this series of 186 patients with paraplegia the level of the lesion was as follows :—

Cervical (including disease the lower part of which involved the cervico-thoracic junction)			16
Thoracic	High .. 22		
	Mid .. 83	.. .. ..	157
	Low .. 52		
Thoraco-lumbar junction	.. .. ..	.. .. ..	12
Lumbar	.. .. ..	.. .. ..	1
			186

Paraplegia with thoracic disease therefore makes up 84·4 per cent of all paraplegia, in this series.

Most writers state that paraplegia is not only commonest in thoracic disease, which is itself far commoner than disease at other levels, but also relatively commoner with disease in the thoracic than at any other level. In the writer's personal series of 92 paraplegias it happened, however, to be relatively commoner with cervical disease (11 paraplegias in 40 patients, i.e., 27·5 per cent) than with thoracic (78 paraplegias in 376 patients, i.e., 20·7 per cent).

DIFFERENTIATION OF POTT'S PARAPLEGIA INTO CLINICAL TYPES

Paraplegia in Pott's disease presents much variability in clinical appearance. It varies in onset. It may be the first sign of the disease either to the patient or his friends. Another patient may have had disease of the spine as a child, leaving him with a kyphus but otherwise strong and well for forty years—and then a paralysis supervenes. The paraplegia varies in degree : sometimes it is complete below a certain level, sometimes only very partial, sometimes asymmetrical on the two sides. The persistence of the paralysis may be for only a few months, or it may be permanent without any tendency to recover under treatment.

No single type of paraplegia is characteristic of Pott's disease. This variability accounts for the divergence of opinion that has existed in the past as to the etiology of the paraplegia, since dogmatic theories of causation advanced by one authority based on an experience of comparatively few cases may be quite unacceptable to another who has seen patients mainly of a different clinical type.

By comparison of a large number of patients with paraplegia it is found, however, that they can be classified as belonging to one or other of certain distinct clinical types. In recent years some rationalization of our ideas upon paraplegia has been attained in this way by Professor E. Sorrel and Mme Sorrel-Dejerine[31] working at the Hôpital Maritime at Berck-sur-Mer. These writers consider that the majority of paraplegias in Pott's disease fall into one or other of two main types. Their first type (A) is paraplegia with onset early in the disease. As far as can be assessed the paralysis supervenes between the sixth and eighteenth month. The progress of the paralysis is rapid and when fully established it tends to be complete. Their second type (B) is paraplegia of late onset, that is to say, it develops at least two years—often many years—after the onset of the disease. It develops but slowly and is seldom complete. They mention also a third type (C), that of transitory paraplegia. In essentials this is a subdivision of Type A, but a mild and ill-sustained paralysis. These writers have sought to show, moreover, that there is a considerable difference in the pathological findings between Types A and B, and suggest that two different clinico-pathological types should therefore be recognized which, they claim, have an entirely different etiology.

Clearly it is of great value to obtain a division of patients by clinical means into distinct types, if such types can be shown to have consistent differences in their etiology, since the only sound basis of treatment is an understanding of the causative factors in each individual case.

The Sorrels' differentiation of "early onset" and "late onset" paraplegia has provided a basis for further investigation and it has been possible to produce a somewhat amplified, but nevertheless simple, classification of patients with paraplegia which seems justified on both clinical and pathological grounds, and which is therefore of value with a view to prognosis and treatment. This classification is as follows :—

Early Onset Paraplegia.—

Type I.—Paraplegia with early active disease : arising, persisting, and diminishing in direct relationship to the activity of the tuberculous infection of the spine. Typically early in onset, usually within the first two years of the disease. Typically a complete paraplegia. Typically recovers completely, if the patient survives.

Type II.—Paraplegia associated in onset with early active disease, but persisting permanently, even if the tuberculous infection in the spine has become completely quiescent. In most cases indistinguishable in early stages from Type I. Typically a complete paralysis at first, but frequently showing some degree of recovery before passing into a state of permanent paralysis.

Late Onset Paraplegia.—

Type III.—Late onset paraplegia appearing at any time up to many years after apparent quiescence of the disease. At the time of onset of the paralysis evidence of activity of the tuberculous lesion may or may not be present but is frequently lacking clinically. Almost always only a very partial paraplegia.

With the passage of time Type III paraplegia is found to fall into one or other of two subdivisions :—

Type III a.—Late onset paraplegia recovering under further conservative treatment as for active tuberculous disease of the spine, whether this is clinically present or not.

Type III b.—Late onset paraplegia not recovering under any form of treatment, whether active tuberculous disease of the spine is clinically present or not.

If the 193 paraplegias in the 186 patients in the series are divided into these types the following figures are obtained :—

Early onset paraplegia—	
Type I	76 (39.4 per cent)
Type II	21 (10.9 per cent)
Late onset paraplegia—	
Type III	96 (49.7 per cent)
(Type IIIa 58 ; Type IIIb 38)	

Reference to the mortality rate from paraplegia has been left until the clinical types have been defined, since the only type which has a large and closely associated mortality is Type I—paraplegia with early active disease. Late onset paraplegia rarely leads directly to a fatal issue. The mortality rate for Type I paraplegia in this series has been approximately 30 per cent.

TYPE I: PARAPLEGIA WITH EARLY ACTIVE DISEASE

THE CLINICAL PICTURE

Recognition of the Type.—This consists of finding early active Pott's disease associated with the paraplegia. Generally Pott's disease has been recognized before the onset of paralysis. Sometimes the paralysis is the first trouble of which the patient complains. Even so, the presence of Pott's disease is generally shown by early angulation, local or referred pain, and X-rays.

Sometimes in adults, in whom bone destruction is slower than in children, disease may be present in the vertebral bodies for some time before there is bone collapse or external deformity, and especially is this so when the disease is confined at first to the posterior parts of the bodies. The best X-rays may then fail to show bone destruction, and it may be months before a lateral film reveals change in the posterior region of a vertebral body.

Disease arising in a lamina or one of the postero-lateral intervertebral joints may likewise give rise to paraplegia without apparent X-ray change. Both are rare, except in suboccipital disease. All such paraplegia without X-ray change is rare, and is indistinguishable from that due to any extramedullary tumour. This will be referred to again later as the ' spinal tumour ' type of paraplegia with early active disease. (*See also* p. 776.)

Onset.—The onset of Type I paraplegia is almost always within the first two years of the disease, usually at or just before the time the spinal disease reaches its fullest development.

Progress.—The progress of Type I paraplegia is usually rapid, being further accelerated by lack of treatment. Muscular weakness, inco-ordination, and spasticity usually appear first, and progress to a paraplegia in extension, with increasingly frequent attacks of general flexor spasm. As the paralysis deepens it may become a paraplegia in flexion. In the worst cases of all—especially if treatment is

neglected, though sometimes in spite of it—much spasticity may be lost and the paralysis may become almost completely flaccid. With such the outlook is always grave; should recovery commence, its first sign will be a return of spasticity. Although motor changes are often the first to appear they are sometimes preceded by changes in sensation. In distribution sensory changes are variable; generally the extremities are affected first and most completely. There is no constant rule as to the order of loss of the various types of sensation. Vibration sense and deep pressure sense are, however, usually the last to disappear. When the paralysis is fully developed there is typically a zone of hyperæsthesia at the upper border of the affected area. Disturbance of sphincter control is often a late feature. Occasionally it is the first sign. Retention of urine and fæces occurs at first. Later automatic action of the sphincters may become established, or, in the most severe cases, the sphincters are completely paralysed and exert no control.

With deepening paralysis much vascular and 'trophic' change may be present in the extremities and in parts exposed to pressure, e.g., the skin of the back, making the avoidance of pressure sores difficult.

A transitory paralysis, lasting for perhaps only a few weeks, and rapid both in onset and recovery, occurs occasionally. It is most frequently seen at an early stage, when the patient is still walking about, and recovery is then frequently simply a response to putting the patient at rest with fixation.

Some degree of disturbance of cord function, as shown by weakness or absence of the abdominal reflexes or by an increase in the tendon reflexes in the legs, occurs in about 50 per cent of children with cervical or thoracic caries at some stage in their illness, but cannot be classed as paraplegia. This is less noticeable in adults.

Very occasionally, with lumbar disease, a lower motor neurone paralysis is seen from involvement of the cauda equina, the paraplegia then being flaccid. A flaccid paralysis of the muscles innervated by nerves arising at the level of the lesion, as a result of the interruption of function of the anterior horn cells at that level, is sometimes seen, though the muscles supplied from a lower level are spastic.

Thus in low cervical disease the arms may be flaccid while the abdomen and legs are spastic. Localized flaccid paralysis of muscles may also arise from pressure by abscesses upon nerves outside the vertebral canal and is seen not infrequently in the intercostal muscles with thoracic disease. A similar disturbance of sympathetic nerves is sometimes seen[1], giving rise to abdominal distension and other upsets of bowel function, but is by no means confined to paraplegic patients.

Recovery.—Type I paraplegia can remain stationary for many months—certainly over a year—and then recover completely. Usually, however, those that recover show some improvement after six months. Recovery is generally associated with—sometimes preceded by—clinical or X-ray evidence that the disease itself is lessening. Pyrexia is less, root pains disappear, or by X-ray bone destruction is seen to have ceased, abscess shadows are smaller, or some recalcification is apparent. Recovery is usually slower than development. Some slight abnormality such as an absent abdominal reflex, an extensor plantar response, or an increase in the knee-jerks, may persist for many months or even years, but on the whole recovery from Type I paraplegia under efficient treatment is, if the patient survives his illness, for all practical purposes complete. Only occasionally does the paralysis of a patient who is under efficient treatment pass from Type I into Type II, remaining permanently after the main activity of the disease is past.

Cause of Death.—The direct mortality from Type I paraplegia is difficult to assess. It cannot be said how many patients who die paralysed would anyway have died from their spinal infection. Death with paraplegia may be due, as sometimes in Pott's disease without paralysis, to generalized tuberculosis or meningitis.

Septic absorption from sores and ascending urinary infections are the two causes of death particularly associated with paraplegia. In Type I paraplegia observed by the writer septic absorption from sores has been the most common cause of death (45 per cent); general spread of tuberculosis the next most common (30 per cent).

Cerebrospinal Fluid.—Some abnormality is always found in the cerebrospinal fluid in Type I paraplegia. All features of Froin's syndrome are not always present, but increase of protein is constant.

Xanthochromia and spontaneous coagulation, which depends upon the presence of fibrin, may be absent. In any case the changes are not characteristic of paraplegia from Pott's disease, being the same as those found with syphilitic myelitis or spinal neoplasm.

THE PATHOLOGY AND ETIOLOGY OF TYPE I PARAPLEGIA

Clinically we have seen that Type I paraplegia arises, persists, and recedes in direct relationship to the activity of the disease itself. We therefore expect that the morbid anatomy will reveal the origin of such paraplegia in factors which can appear and later disappear.

The usually accepted explanation of Type I paraplegia is that the cord is compressed mechanically by an 'abscess'. In the majority of fully developed paraplegias of this type an element of compression certainly is present, though the compressing factor, as we shall see, is frequently granulation tissue rather than fluid pus as would be suggested by the term 'abscess'.

Type I paraplegia may, however, occur without true compression of the cord and its meninges. There must therefore be other factors which can alone produce interference with cord function in such cases, and which are worthy of most careful consideration, for such factors may be present also in others in addition to an element of mechanical compression, sharing in the production of the paralysis and providing something more than a straightforward mechanical basis for the paraplegia, which will not therefore necessarily be relieved completely by mechanical means.

The non-mechanical factors thus concerned in Type I paraplegia are the vascular and toxic reactions in the cord secondary to the neighbouring active tuberculosis, and these will be considered before passing on to the more obvious element of mechanical compression.

Type I Paraplegia without Mechanical Compression of the Cord.—The following is a case report of this condition:—

Case 1.—A boy, A. C., aged 5, was admitted to Pyrford Orthopædic Hospital in May, 1931, with a mid-thoracic kyphos and a history suggesting tuberculous disease for four months. After nine months on a spine frame he developed bronchopneumonia and died. For the last six weeks he had increasing paralysis, and at the time of death he had a spastic paraplegia in extension with sensory loss to the 6th thoracic segment and loss of sphincter control. During life, X-rays suggested that the 5th to the 8th thoracic vertebræ were infected and largely destroyed.

A sagittal section of the spine is shown in *Fig.* 517. There is infection of seven vertebræ —not four as was thought during life—and of all intervening discs. It is indeed common—

especially in children—to find at post-mortem that more vertebræ are infected than is shown by X-rays during life. Tuberculous granulation tissue has spread on all surfaces of the vertebral bodies. It forms a large mass anteriorly, and behind—opposite the main area of bone destruction—has invaded the extradural space. Here a rounded mass lies in front of the dura mater and has spread around both sides to form a mass behind the cord also. There is much caseation, and in the central area of destruction is a small abscess containing fluid pus. There is no fluid pus in the extradural space, but here the granulation tissue contains many small areas of caseation. The surrounding tissue reaction is intense. All the soft parts on the front and sides of the spine are œdematous, including even the fat in the posterior mediastinum. This œdema extends, moreover, for the whole length of the specimen. The extradural space in the central region of the specimen is filled, partly by granulations, partly by œdematous fat and connective tissue. Vascular engorgement is marked, especially below the lesion. The small vessels are distended even on the walls of the aorta. The vessels on the cord itself are congested, especially below the main site of disease. The dura mater is intact. Its inner surface is smooth, shining, and normal in appearance.[20] Its outer surface is adherent to the epidural granulations, but the membrane itself is not apparently changed macroscopically. With but slight difficulty the granulation tissue can be 'peeled off' the dura mater, leaving the latter apparently normal.

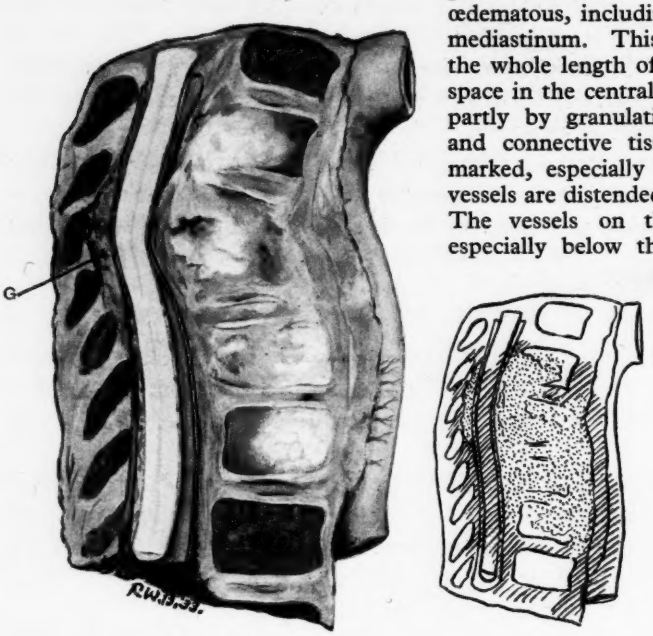


FIG. 517.—*Case* 1. Type I paraplegia without true mechanical compression of the cord, with early mid-thoracic Pott's disease, in a boy aged 5. Tuberculous granulations have invaded the extradural space and have spread round to the posterior surface of the dura (G). The soft parts of almost the whole specimen are œdematous. In B the area of vascular engorgement (shaded red) around the actual area of tuberculous infection (stippled) is emphasized.

Here, then, is a specimen from a patient with Type I paraplegia in which there is no true mechanical compression of cord tissue. Tuberculosis has invaded the extradural space and surrounds the dura mater, but the cord itself is not pinched or distorted in any way. What then is the cause of the paraplegia? *A study of specimens from patients with Type I paraplegia reveals only one constant factor—the spread of active tuberculous granulation tissue to the epidural space, where it comes into intimate contact with the meninges.*

Spread of infection to the extradural space may be early, especially with infection commencing in the posterior part of a vertebral body, and is but little checked by the posterior common ligaments, which are wide only behind the intervertebral discs.

Granulations appearing in the anterior part of the extradural space spread round one or both sides of the dura mater and up and down the canal, often finally forming a ring—or rather tube—of infection about the cord. In the granulations small areas of caseation and pus formation appear, but rarely in the early stages of infection is there a collection of fluid pus in the extradural space which truly deserves the term of 'abscess'. The dura mater is found covered with a layer

of shaggy granulations, and at first sight it appears that there is a true tuberculous 'pachymeningitis'. This is the term used to describe this picture in many of the older case reports,[4, 19] but it is misleading, since the dura mater is not affected by tuberculosis but is acting as an excellent barrier to its spread.

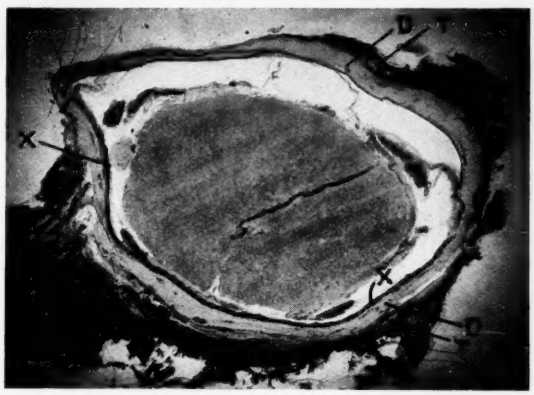


FIG. 518.—*Case* B.H.T. 3. Type I paraplegia without compression of the cord. Low-power magnification of a section of the cord and meninges from a man, aged 61, who died in St. Thomas's Hospital with a Type I paraplegia of four months' duration with mid-thoracic Pott's disease. The relationship of the extradural granulation tissue (T) to the intact dura mater (D) is shown. Hyperplasia of the dura mater is shown (X X) where thickening is taking place with appearance of fibroblasts and small round cells on its inner surface. The sharp line of demarcation between tuberculous granulations and intact dura mater is everywhere evident.

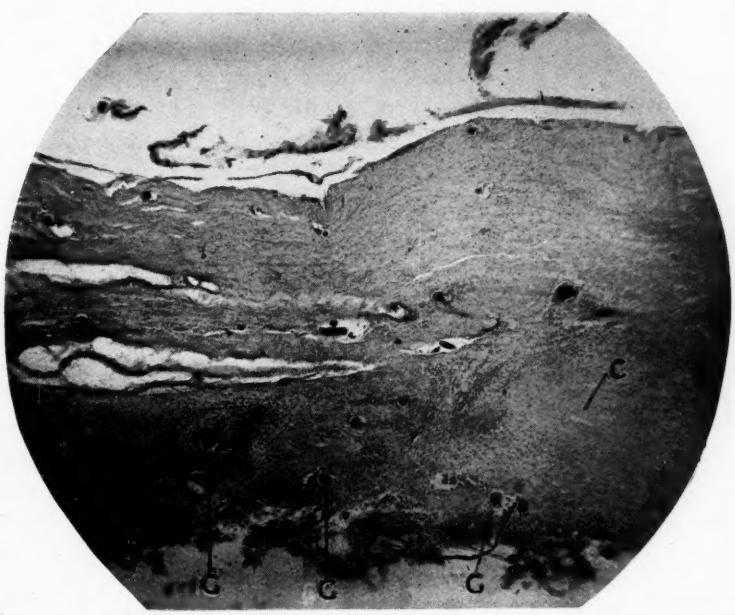


FIG. 519.—*Case* B.H.T. 3. Higher magnification than *Fig.* 518 to show the relationship of the tuberculous granulation tissue (below) to the dura mater (above). The two can be easily peeled apart, as to the left of the section. The dura mater shows some thickening and vascular engorgement. In the tuberculous tissue are many giant-cell areas (G) and caseation (C).

It is true, as shown in the photomicrographs *Figs.* 518, 519, that the dura mater may be thickened where it is in contact with granulation tissue, but this is due to the growth of young connective-tissue cells outside the endothelium on the

inner surface, so that the barricade is, as it were, constantly strengthened from within. The correct description is that there is an extradural spread of tuberculosis with some reactionary hyperplasia of the dura mater.[35, 7, 24] With care the granulations may be stripped from the dura mater, leaving it of normal integrity and appearance.

FIG. 520.—Type I paraplegia. The cord exposed from behind and the dura opened. The cord in the centre of the figure is lying anteriorly in a bed of tuberculous granulation tissue. The dura mater is intact. Engorgement of all the vessels on the cord, especially below the disease, is well shown. (*By permission of Mr. H. J. Seddon.*)

If, then, paraplegia can occur in the presence of epidural tuberculosis but in the absence of both mechanical compression of the nervous tissue and of infection of the dura mater, we must seek yet other factors as the main cause of the paraplegia. In the above description of a specimen from a patient dying with Type I paraplegia the widespread toxic and vascular-tissue reaction as shown by œdema and vascular engorgement has been stressed. There is nothing unique about this reaction as far as disease of the spine is concerned. It occurs about every active tuberculous focus. It commences as soon as the focus is established, increases as the disease reaches its height, and disappears with its recovery. It is marked with spinal disease because spinal disease is often a large and, in its early stages, an ill-limited focus of tuberculosis. Once the infection has passed its acute stage and some general and local immunity has been established, this surrounding reaction largely disappears and is comparatively little in evidence about a chronic focus.

In considering the tissue œdema and the vascular engorgement it is hard to say with certainty how far these are interdependent factors. The œdema appears to be distributed all round the lesion equally and is probably largely a local toxic or allergic change.

The vascular engorgement is more definite below the main centre of infection, and is probably due mainly to obstruction of the veins by pressure of the surrounding œdema and of the invading tuberculous tissue (*Fig.* 520). The cord, lying in the centre of the area of intense tissue reaction and separated from it only by the dura mater, undoubtedly shares both in the œdema and the vascular changes. With regard to the latter, it must be remembered that the cord below the uppermost cervical region depends for its circulation upon small segmental vessels which reach it by passing in along the nerve-roots from vessels in the extradural space. These latter, both arteries and

veins, are connected with branches from the vertebral, ascending cervical, intercostal, lumbar, and ilio-lumbar vessels.[12]

Should the vessels of the extradural space be congested, compressed, or destroyed over a wide area, there is no doubt that the circulation in the cord must be greatly affected. A sudden onset of complete paraplegia may in fact occur as a very rare disaster from an acute thrombosis of the vessels in the cord which has spread directly from an extensive thrombosis in extradural vessels. Such a case is described by Seddon (p. 774).

There must also be much destruction of the lymphatic vessels, which are numerous in the extradural space and send branches along the nerve-roots which are believed to be connected directly with the intercellular spaces in the cord itself.

Let us now see what microscopic changes are present under these conditions within the cord.

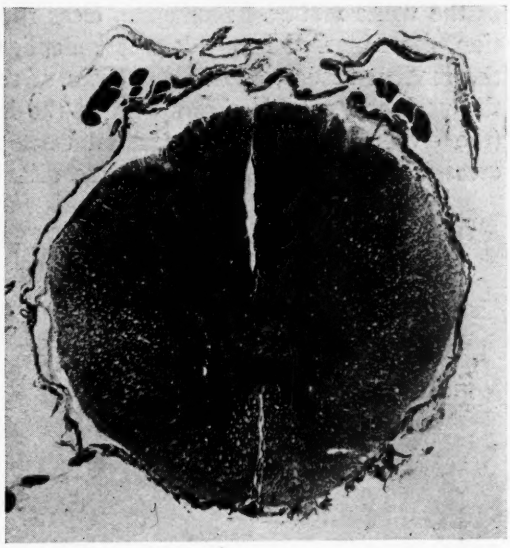


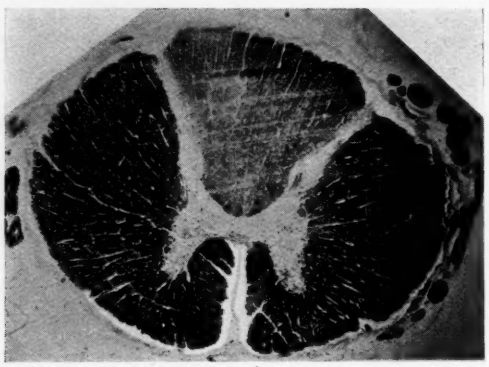
FIG. 521.—*Case* B.A.N. 1. Type I paraplegia without compression of the cord. Section of the cord at the level of the disease from a woman aged 58 with early mid-thoracic Pott's disease admitted to the National Hospital, Queen's Square, with a paraplegia for six weeks. Tuberculous granulation tissue had invaded the extradural space, but there was no mechanical compression of the cord. The appearance of fenestration in the large myelinated tracts of the white matter is well shown. (*For this and the following illustration the writer is indebted to Dr. J. G. Greenfield, Pathologist to the National Hospital.*)

In *Figs.* 521–523 we see sections of the cord from two patients both dying with complete paraplegia of Type I in association with thoracic caries. In both patients there was much extradural tuberculosis, but in neither was there compression of the cord by granulation tissue or pus. In both the general histological features are similar. Where the dura mater is in contact with granulation tissue it is slightly thickened—partly by œdema, partly by new connective-tissue formation on its inner surface. Young connective tissue and round-celled infiltration are present about the meningeal vessels. The arachnoid membrane appears normal and the subarachnoid space is distinct. In the nerve-roots and on and in the cord itself there is some increase of young connective tissue and round cells about the vessels, but this must be regarded simply as a reaction to the

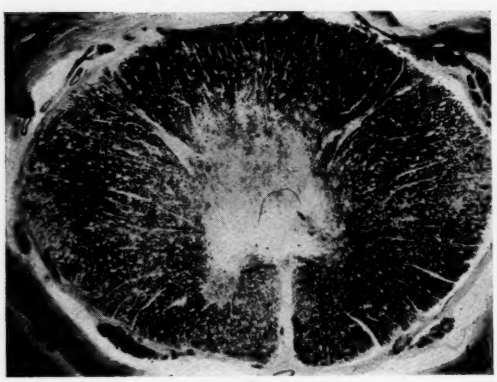
FIG. 522.—*Case* B.A.N. 1. Type I paraplegia without compression of the cord. High-power magnification of *Fig.* 521 to show the rounded spaces which give the fenestrated appearance to the white matter of the cord. These appear to be degenerating and distended myelin sheaths, and in many a swollen axis cylinder still persists (X).

extradural changes, for it is very different from anything seen in the characteristic zone of infection outside the dura.

In the cord, at the level of the lesion, is one striking change—most noticeable in the white matter. Numerous clear round areas give an appearance of fenestration, most marked in the lateral funiculi, in the anterior fasciculi on either side of the median fissure, and in the bases of the posterior funiculi, as well as all round the periphery. This shows best in the lateral and anterior cerebrospinal fasciculi (the crossed and direct pyramidal tracts). These clear areas are apparently the cross-section of fusiform or rod-like distensions of degenerate myelin sheaths. In



A



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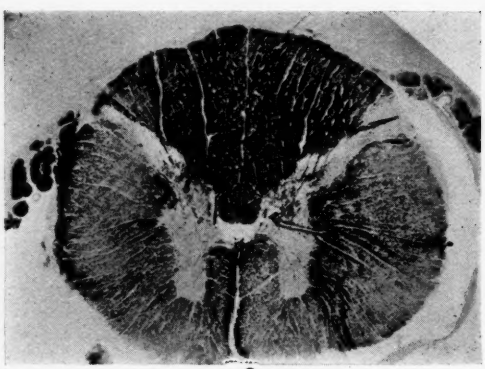


C

FIG. 523.—Type I paraplegia. Sections of the cord from a woman aged 43 who died in St. Thomas's Hospital with a paraplegia of only six weeks' duration from early mid-thoracic Pott's disease. Stained by the Weigert-Pal method to show degeneration of white matter at the level of the disease and in ascending and descending tracts above and below. A, Two nerve-roots above the disease; B, At the level of the disease—the appearance of fenestration in the large myelinated tracts is clearly visible; C, Two nerve-roots below the disease. Note that the appearance of fenestration is present only at the level of the lesion.

some of them swollen and degenerating axis cylinders are visible (*Fig.* 522), in others these have disappeared. In a longitudinal section these rod-like spaces appear at intervals in the sheaths, the continuity of the myelin breaking abruptly at either end or fragmenting into poorly staining débris. The exact significance of these distended myelin sheaths is obscure, but, since they occur only at the level of the lesion, they must have some other origin than the simple ascending and descending degeneration of myelin sheaths which extends rapidly above and below (*Fig.* 523). The grey matter shows some degree of perivascular infiltration

and perhaps some unusual preponderance of glial tissue, but the nerve-cells appear little altered.

That these changes have arisen as part of the general toxic and vascular reaction about the active tuberculous focus seems certain. Any more exact definition of the changes and of their mode of origin seems impossible.

Charcot[4] and Michaud[21] described the cord change as a myelitis—implying that it was actually tuberculous, which it certainly is not. Ménard[20] considered that it was a " simple œdema ", and Zeigler[42] that it was due to obstruction of afferent vessels. Strumpell[35] thought the lesion primarily a degeneration of nerve tissue with a reactionary phagocytic round-celled infiltration and œdema. Schmaus[28] described it as a toxic œdema. Fickler[9] based everything on lymphatic obstruction, and recently Hassim[17] appears also to have favoured this. A very similar appearance with vacuolation in the white matter as described above was produced by Kahler[18] in 1882 by introducing non-infective substances such as beeswax and laminaria into the extradural space, and was discussed by Schmaus.

Whatever other factors, mechanical or otherwise, may contribute to the paraplegia, these toxic and vascular changes in the cord will have to be considered in all cases where the paraplegia is associated with active tuberculosis in the immediate neighbourhood of the cord. They have therefore been described at length, as their importance is undoubted.

It is clear that we have here seen enough cord change to explain such interruption of function. The change, moreover, is of a type which may well allow of much recovery of function when the neighbouring infection—to which it is a response—itself recovers.

The pathological picture agrees with our clinical knowledge of the outcome of Type I paraplegia. The paralysis will generally recover on conservative treatment if the patient lives long enough for his tuberculosis to heal.

Mechanical Compression of the Cord as an Additional Factor in Type I Paraplegia.—The occurrence of Type I paraplegia without mechanical compression of the cord has been stressed, since in considerations of treatment the presence of some mechanical factor is so often taken for granted as being present always and present alone.

That some slight pressure is in all cases exerted upon the dura by the granulation tissue, with obliteration wholly or in part of the subarachnoid space, might be thought likely from the differences in the cerebrospinal fluid above and below the focus. That the normal circulation of the cerebrospinal fluid is interrupted is certain from such evidence as Queckenstedt's test and the subarachnoid injection of lipiodol above the lesion, as well as from the chemical changes. The chemical changes are variable, though one—the increase of protein, mainly as globulin—is constant. It is possible, as Greenfield and Carmichael[16] have pointed out, that this last change may be secondary to increased filtration from the vessels from engorgement below the lesion rather than to obstruction to the flow of fluid. Even if the " cavité close " theory of Sicard and Deschamps is true, the obstruction in the subarachnoid space may be due in part at least to hyperplastic thickening of the dura mater and to swelling of the cord itself.

Mechanical compression of the cord is usually present in Type I paraplegia, and such compression may be gross. When compression is by granulation tissue it may be a slight general constriction, the cord being then actually larger than

normal immediately above and below from œdema, or there may be distortion from a mass in front or to one side (*Fig.* 524).

When compression is by the fluid pus of an abscess it is frequently very marked, as in *Figs.* 525, 526. In the two specimens illustrated certain features are similar. In neither is the bony canal narrowed; in both the epidural space is filled by tuberculous tissue and pus; in both the compressing extradural abscess is but a process of a larger paravertebral abscess pushed back through the main areas of destruction; in both the tension in the tuberculous epidural tissue and in the cord itself must therefore have been the same as that in the paravertebral abscess.

In the writer's experience all specimens showing compression by fluid extradural pus show also that this is an extension from a larger paravertebral abscess. In this the writer agrees with the Sorrels,[31] but he disagrees with their statement that such connection with a paravertebral abscess acts as a safety-valve, preventing high tension in the part of the abscess immediately related to the cord.

The Sorrels' view is probably correct in cervical and lumbar disease, where a paravertebral abscess can spread easily between muscle planes and sinus formation may result, but in the thorax such abscesses are thick-walled, localized, and fixed, and any surgeon who has drained such an abscess by costotransversectomy knows that its contents are under high tension.

Actually where an epidural abscess is in continuity with an extravertebral abscess within the thorax the tension in the contents must be rising and falling with the respiratory movements, and such changes of tension must be transmitted direct on to the cord like the blows of a water hammer. The extension of a paravertebral abscess to the extradural space must therefore increase the chance of paraplegia on mechanical grounds. Clinical findings corroborate these suggestions, for although almost all patients with thoracic caries develop an extravertebral abscess, sinus formation is rare. It has long been recognized that should a patient with paraplegia from thoracic caries develop a sinus and thus establish drainage naturally the paraplegia will recover rapidly.[20] It is of much interest to note that Percivall Pott, writing his first tract upon palsy of the lower limbs in 1779, says that he was particularly led to consider the condition by the care of a boy who had a " restoration of the use of the limbs immediately after a seemingly accidental abscess near the part ".

The extramedullary tumour type of paraplegia in which early disease at the back of a vertebral body is not shown by X-rays is clearly a matter of ill chance. Disease has arisen where it can immediately affect the cord.

To summarize the pathology of Type I paraplegia, we may say that it is due to toxic and vascular reaction in the cord, supplemented in many cases by compression by granulation tissue or pus.

Mild and transitory Type I paraplegias are probably due to toxic and vascular cord changes only, without any great element of true compression.

In what percentage of Type I paraplegias the compression factor is gross it is impossible to state. In most of those that die with a severe paralysis it is well marked. Even when it is severe, however, it would seem likely that drainage of pus from a compressing abscess, either spontaneously or as the result of operation, or the rapid recovery of an active tuberculous focus with disappearance of granulation tissue, might well allow of restoration of cord function as long as the compression had not been too long maintained.

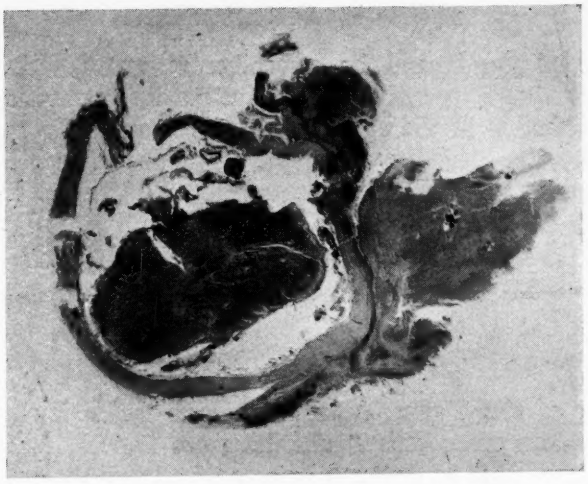
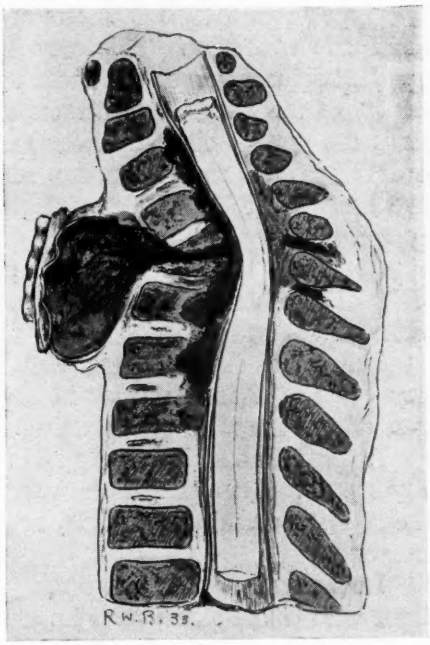


FIG. 524.—*Case* B.E.H.4. Type I paraplegia with compression of the cord. Section of the cord and meninges from a woman aged 52 with severe Type I paraplegia from low thoracic Pott's disease who died in St. Thomas's Hospital. At post-mortem the extradural space was full of tuberculous granulation tissue and semi-fluid caseous material compressing the cord. The cord is seen distorted, and masses of granulation tissue are seen in some areas adhering to the outer surface of the dura mater, which has, however, acted as a barrier to its spread. (The fragmentation of the dura mater is an artefact.)



FIGS. 525, 525.—Type I paraplegia with true mechanical compression of the cord.

Fig. 525 shows the upper part of the spine of a child aged 4. The cord is surrounded by granulations and caseous matter which are in continuity anteriorly through the main site of destruction in the upper thoracic region with a thick-walled paravertebral abscess containing fluid pus. (*Specimen No.* 16.B.2, *Museum of the Medical College, University College Hospital.*)

Fig. 526 is another specimen showing a similar state of affairs with cervical disease. (*Specimen No.*153, *Museum of Pathology, St. Thomas's Hospital.*)

TYPE II PARAPLEGIA

Type II paraplegia, it will be remembered, was described as paraplegia associated in onset with early active disease but persisting permanently, even when the tuberculous infection in the spine has become completely quiescent. Should the patient survive, the majority of paraplegias with early active disease recover under treatment as the disease itself recovers (Type I). We have seen what pathological factors may produce the paraplegia and yet be capable of resolution to allow of this recovery. A certain small number of paraplegias which have their onset with early active disease remain to a large extent permanently paralysed (Type II).

THE CLINICAL PICTURE

Recognition of the Type.—Most paraplegias which finally turn out to be of Type II are at their onset indistinguishable from Type I. It is, however, right to place such cases as soon as they can be recognized in a separate class, since it is clear that some further pathological factors must have appeared to make recovery impossible.

Occasionally a dramatically sudden onset of profound paralysis may suggest the occurrence of some major disaster, mechanical or vascular, and will lead us to expect at the start that the cord will subsequently prove to be irretrievably damaged (*see* Seddon's article, p. 773).

Type II paraplegia is uncommon. Most patients in whom it occurs have had inadequate treatment. This may have taken the form of real neglect, as when a patient refuses proper treatment or is unable to obtain it in the stage of greatest activity of the disease, when bone destruction is occurring rapidly. On the other hand, the patient may be under treatment with good fixation but becomes so ill or develops such severe pressure sores that ideal treatment has to be jettisoned to save life. Most Type II paraplegia is, therefore, the result of a failure to treat Type I paraplegia properly.

As would be expected, since efficient splintage during the destructive stage of the disease has been lacking, Type II paraplegia is almost always associated with great deformity.

Before the stage of permanent paralysis is reached some degree of recovery may occur. Though it may be extensive, the spasticity and motor paralysis is then often somewhat asymmetrical.

Anæsthesia may be patchy and irregular, and control of one or both sphincters may return. This partial recovery has usually occurred at the time when the disease was becoming quiescent, i.e., at the time when Type I paraplegia recovers completely.

In the ' permanent ' stage the patient can often live fairly comfortably, getting about with calipers and crutches or sticks, but he is rarely fit for work except in an institution with special accommodation for cripples.

In some, in whom the spinal lesion has been very extensive, infection may grumble on for years, occasionally becoming manifest by some exacerbation, which is then sometimes accompanied by a further deepening of the paralysis. In some, therefore, the paraplegia is associated with continued activity. In some there is no evidence at all of persistence of active disease.

PATHOLOGY AND ETIOLOGY OF TYPE II PARAPLEGIA

In any irrecoverable paraplegia it is clear that at a late stage there will be cord degeneration. What, however, are the factors which lead to this degeneration and which turn a recoverable Type I paraplegia into an irrecoverable Type II?

Firstly, it is certain that many Type II paraplegias are established because disease lasts so long before healing and its effects upon the cord, toxic, vascular, or mechanical, are so profound that the damage to nerve tissue, reparable at first, becomes permanent. Considering the degree of cord change in Type I paraplegia it is surprising that Type II is not more common. This is the paraplegia from long-continued disease.

As a matter of fact, profound Type I paraplegia in flexion, implying involvement of all tracts of the cord, which lasts for more than a very few months rarely recovers. Likewise, a paraplegia which becomes flaccid—though spasticity may return—always appears to be irrecoverable. Fortunately, both are rather uncommon in paraplegia with early active disease. Permanency of paraplegia is not closely dependent upon length of persistence of the paralysis; but rather upon the type and time factor combined. One very rare cause of a sudden onset of irreparable cord damage in early disease is an acute thrombosis spreading into the vessels of the cord from without. An example is described by Seddon.

That a mechanical factor—pressure upon the cord by bone—may sometimes be the underlying factor in Type II paraplegia is made likely by the fact that splintage of the spine has often, for one reason or another, been lacking at the time of most rapid bone destruction, and by the consequent gross deformity which is generally present.

That compression by bone is a common cause of paraplegia has been long disproved, and up to recent times some authorities have even asserted dogmatically that it never occurs. Recently, however, sufficient examples have been described in detail to convince even the most hardened theorists that compression by bone is occasionally present.

The most terrible kyphos may occur without paraplegia. As long as the posterior vertebral arches with the articular processes and posterior intervertebral joints are intact, destruction of the bodies with a falling together in front results only in a hinging of the vertebræ, one upon another, behind. There is a shortening of the vertebral canal but by no means necessarily any loss in its diameter; and a sliding apart of the laminæ may actually increase the capacity of the canal posteriorly. Compression of the cord by bone may, however, occur in Pott's disease in two ways:—

1. If the posterior intervertebral joints or the pedicles and articular processes are destroyed, *a pathological dislocation of the spine may take place with pinching of the cord.*

2. If destruction of the vertebral bodies leads not only to the removal of bone but to the formation of *loose sequestra or masses of débris*, these may be forced back by the collapse of the other bodies into the vertebral canal, where they compress the cord.

Pathological Dislocation.—This is comparatively common in suboccipital and atlo-axoid disease, where the posterior intervertebral joints are involved early and massive loss of bone is characteristic. Dislocation in this region is a classical

cause of sudden death in Pott's disease (*Fig.* 527). Dislocation at a lower level is uncommon. In the thorax, following destruction of the posteriory intervertebral joints, the upper spinal segment either falls down anteriorly in front of the lower, in which case the cord is pinched over the upper end of the lower segment (*Fig.*

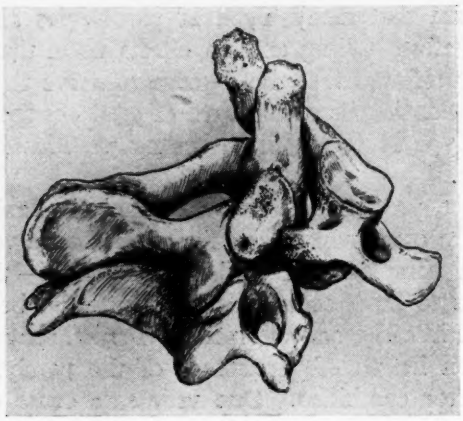


FIG. 527.—Atlo-axoid dislocation. Showing the element of rotation usually combined with the antero-posterior dislocation at this level. Massive destruction of the posterior arch of the atlas is also typical. (*Specimen No.* 3868.1, *Museum of the Royal College of Surgeons.*)

528, A), or slides downwards and backwards over the lower segment, in which case the cord is pinched by the lower end of the upper segment (*Fig.* 528, B).

Two examples of dislocation in which the upper segment has fallen downwards and forwards over the lower are shown in *Figs.* 529–532. The first

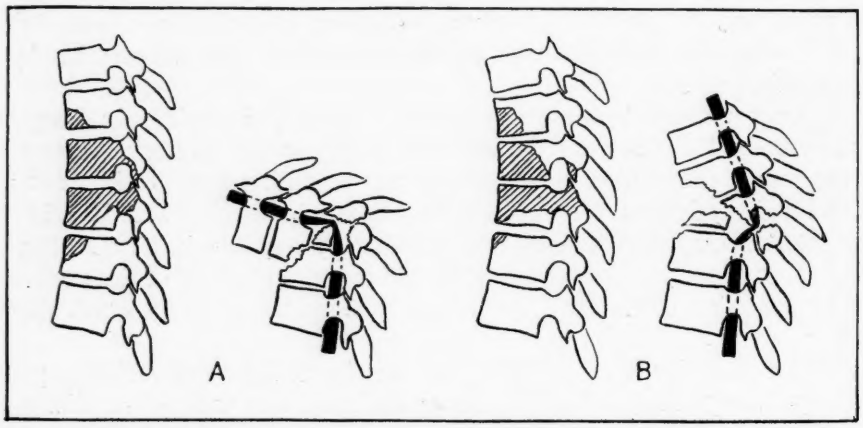


FIG. 528.—Diagrams showing the two ways in which pinching of the cord may result from pathological dislocation from Pott's disease in the thoracic region. The shaded areas are those in which bone is destroyed. A, The upper segment of the spine has slipped downwards and forwards (as in *Figs.* 529, 530); B, The segment has slipped downwards and backwards (as in *Fig.* 533).

(*Figs.* 529, 530) is from a man aged 33 under the care of Dr. B. Armstrong at the Royal Sea Bathing Hospital, Margate. The case is reported by Seddon (*Case* S.90). He developed a paraplegia while under treatment for sacro-iliac tuberculosis. This became complete, and he died thirteen months after the onset from generalized

FIG. 529.

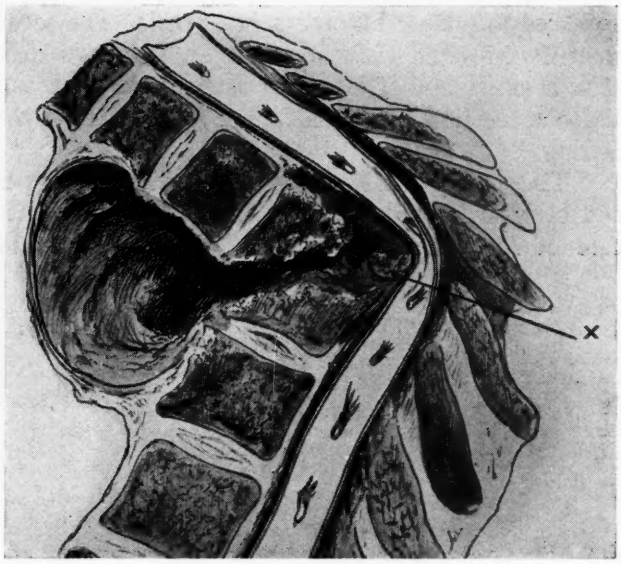


FIG. 531.

FIG. 530.

FIG. 532.

FIGS. 529–532.—Two specimens showing pathological dislocation of the thoracic spine in Pott's disease, with pinching of the cord. In each case the upper segment of the spine has fallen downwards and forwards over the lower, so that the bone compressing the cord is the persisting postero-inferior corner of a vertebral body which has remained in position, forming the apex of the lower segment. *Figs.* 529, 530 are from a case (*Case* S. 90) reported by Seddon, to whom the writer is indebted for the illustration. The laminæ and pedicles were removed during dissection of the specimen. *Figs.* 531, 532 are from Specimen No. 155 in the Museum of Pathology, St. Thomas's Hospital.

tuberculosis and sepsis from sores. During life caries of the 5th to the 7th thoracic vertebræ was recognized, and by X-rays the body of the 6th thoracic vertebra appeared to project into the spinal canal. The second specimen (*Figs.* 531, 532) is from the Museum of Pathology at St. Thomas's Hospital. The presence of a large abscess cavity and of much tuberculous tissue shows that the disease was early and active at the time of the dislocation, but details of the case history are unfortunately lacking.

The example of dislocation in which the upper segment has slid downwards and backwards over the lower, shown in *Fig.* 533, is from a boy aged 16 under the care of Mr. St. J. D. Buxton. The case is reported by Seddon (*Case* S.62).

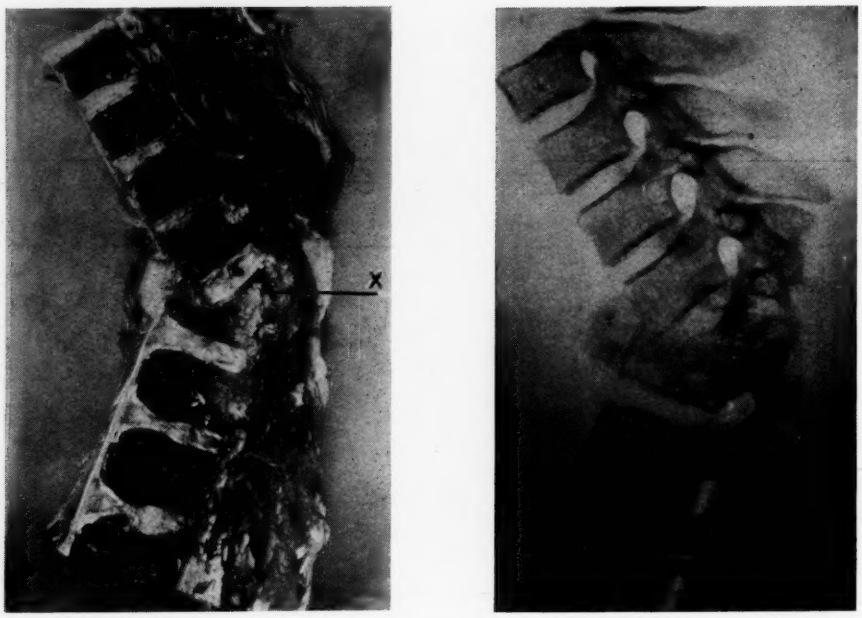


FIG. 533.—Pathological dislocation of the spine in Pott's disease, with pinching of the cord. The upper segment of the spine has slipped downwards and backwards on the lower. The bone compressing the spine is the persisting postero-superior fragment of a vertebral body which has remained in position as the lowest point of the upper segment. From a patient (*Case* S.62) reported by Seddon, to whom the writer is indebted for the illustration.

Mid-dorsal caries was recognized in October, 1928, and paraplegia commenced in November of the same year. This became complete, and in January, 1929, he was transferred from a sanatorium to King's College Hospital. Laminectomy was performed, extradural abscesses were emptied, and the extradural space was drained for three days. He died in April, 1929, from generalized tuberculosis and sepsis from sores, without recovery of the paraplegia. In all these three patients the mechanism of the pinching of the cord by the pathological dislocation is made clear by the specimens and X-rays.

While discussing compression of the cord from pathological dislocation it is worth while remembering that simple laminectomy for a Type I paraplegia has occasionally been followed by a sudden increase in the depth of the paralysis, which has remained permanently. The most probable explanation is that removal of the

only remaining stable part of the spine has allowed dislocation to occur. The writer has seen two such cases, and others are quoted by Seddon.

Pressure by Loose Sequestra or Masses of Débris.—That compression of the cord by sequestra occasionally occurs is well recognized and has been described by Sorrel[31] and others. The mechanism of the introduction of loose sequestra into the vertebral canal by the crushing down of the bodies in front is easy to understand.

A specimen illustrating this disaster is shown in *Fig.* 534. This specimen is preserved in the museum of the Hôpital Franco-Américaine at Berck-sur-Mer and is shown by courtesy of Dr. Jacques Calvé. The patient was the victim of a paraplegia which in spite of treatment was complete and showed no signs of recovery.

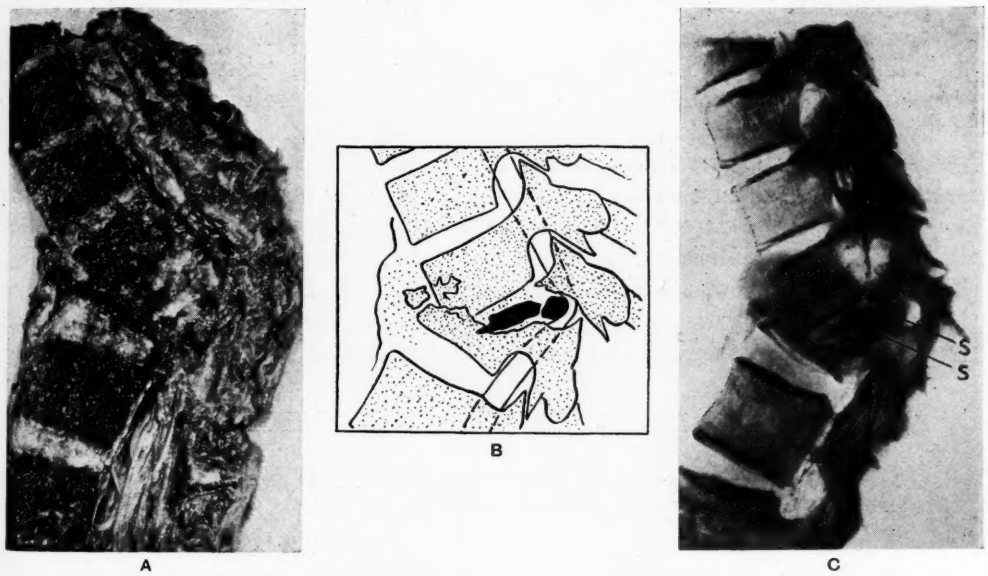


FIG. 534.—A, Specimen showing compression of the cord by displaced sequestra in the Museum of the Hôpital Franco-Américaine, Berck-sur-Mer; B, Diagram of manner of displacement; C, X-ray of specimen, showing the sequestra (S). (*By permission of Dr. Jacques Calvé.*)

At the post-mortem examination it was found that two loose sequestra had been pushed back into the vertebral canal and were compressing the cord.

One rare cause of a very sudden and profound paraplegia coming on with early active disease is what may well be called 'concertina collapse' of a vertebral body. In this an infected vertebra suddenly gives way with expulsion of the bone débris, tuberculous granulation tissue, and pus into the vertebral canal as a semi-solid mass, so that the cord is suddenly and violently compressed. It would seem correct to class this as Type II paraplegia, for it is unlikely that it could ever recover. No example of 'concertina collapse' occurred in the combined series of 193 paraplegias on which this paper is based, and the condition would appear to be rare. A specimen illustrating this has, however, since come into the possession of the writer and is described by Seddon in his paper (p. 773). The condition has also been described by Girdlestone.[13]

While discussing compression by bone it is as well to point out a fallacy which

may arise in the interpretation of some specimens. It is not uncommon, especially in irrecoverable late onset paraplegia, to find a flattened and attenuated cord stretched over a consolidated mass of débris which forms the anterior wall of the vertebral canal at the site of angulation. At first sight degeneration of the cord would appear to have followed compression by this mass. Actually the vertebral canal is often quite spacious behind the cord. The bony canal has been shortened by the angulation. Were the cord of normal length and elastic consistency it would lie free and uncompressed in the undiminished canal (as in *Fig.* 538).

There is no doubt that in such cases the primary factor is cord atrophy (the cord has had to take on more than it can stand), and that with atrophy the cord has become shrunken so that it is drawn forward by its loss in length to lie in close apposition with the anterior wall of the canal. Such a conception is of importance in interpretation of the pathological picture with a view to considerations of treatment of similar cases.

Clinically it may be difficult to recognize true compression by bone. X-rays taken during life frequently fail to demonstrate the presence of sequestra, and may be hard to interpret as to the presence or absence of minor degrees of dislocation. X-rays following subarachnoid injection of lipiodol are disappointing in differentiating the exact cause of the paralysis. The lipiodol may be held up with gross kyphos formation even without paraplegia, and it is just in those, the cause of which cannot be told clinically, that such injection seems to give the most variable shadows and therefore the least exact information.[32] Permanent compression of the cord by annular constricting bands or masses of fibrous scar tissue from healed tuberculosis has been described, but has not been met with by Seddon or the writer.

TYPE III: LATE ONSET PARAPLEGIA

Type III is late onset paraplegia, which may appear at any time up to many years after the apparent quiescence of the disease.

THE CLINICAL PICTURE

The outstanding characteristics of Type III paraplegia are the late and gradual onset, the incompleteness of the paralysis, and the high percentage of permanent paralysis in comparison to Type I.

Time of Onset.—Very variable, a few months to many years after apparent quiescence of the disease. The writer has seen one patient in whom the onset was forty years after her disease was ' cured '.

Development.—Always more gradual than Type I. Muscular spasticity usually the first sign. Sensory change may even be lacking throughout, but there is usually widespread but incomplete anæsthesia. Seldom are the lower limbs completely devoid of slight voluntary power, even at the height of the paralysis. Loss of sphincter control is inconstant. Frequently there is loss of bladder control though the bowel is normal. Occasionally complete double incontinence is seen. Vascular and trophic changes in the skin are very uncommon. Even at its height late onset paraplegia is typically incomplete.

Progress and Response to Treatment.—Late onset paraplegia is not always permanent as suggested by the Sorrels. Under treatment by fixation in recumbency as for active tuberculosis of the spine some recover completely and many

improve, though it is true that the prognosis as far as recovery of the paralysis is concerned is not as good by any means as in Type I. Recovery of late onset paraplegia appears to be more likely in the younger patients.

The combined 96 cases of late onset paraplegia of Seddon and the writer show recovery on conservative treatment in 74 per cent of patients under 16 years old, whereas only 52 per cent recovered over that age.

If recovery of late onset paraplegia sometimes occurs with conservative treatment as for active tuberculosis of the spine, it would seem likely that such activity is present in these patients and, as in Type I, is the causative factor in the paralysis. If this were so, then we would look carefully for signs of such activity, since we would hope that by treating the spinal disease we would be able to cure the paralysis. Actually clinical evidence of activity of the disease in the spine is generally hard to obtain in late onset paraplegia, but this does not mean that active disease is absent. The real difficulty may be summed up by saying that recurrent or chronic tuberculosis of bones and joints may produce very little general or local reaction, while bone destruction and fluid abscess formation may be negligible and pain completely absent.

Pyrexia may be present, but we must exclude tuberculous or other chronic infections in the chest and renal system. X-rays may be indefinite as evidence of activity in the lesion. Extreme kyphos formation makes the degree of bone fusion very hard to assess; actually it is usually less, especially in children, than the X-ray suggests. Calcified abscess shadows may obscure the lesion, but a shadow suggesting a fluid abscess is valuable evidence of activity when it is present. Progress in bone destruction or kyphos formation since a previous X-ray implies further activity at some time, but not necessarily at the time of examination. Pain at the level of infection is suggestive of activity.

Examination of the cerebrospinal fluid in late onset paraplegia is not a very dependable test, since we do not know what factors, apart from active tuberculosis, may or may not be present as the result of the old disease and may produce obstruction in the flow of the fluid. Often the protein in the fluid is not raised to the same degree as in Type I paraplegia. Examination of the fluid in a short series of late onset paraplegias shows that those with the higher figures for the protein content recovered better on treatment than the others, so that the raised protein is perhaps here associated with vascular engorgement from the proximity of active disease rather than with any other factor. The series (*see* Table, p. 760) is, however, too small to be dogmatic in this respect.

As a matter of fact clinical evidence of activity of the spinal focus, though most commonly found in those patients who recover, may be present with some late onset paraplegias which remain permanent, and again is sometimes unobtainable in those that recover, so that it is far from reliable in making a prognosis.

The only real test of ability to recover is to put the patient under treatment by fixation in recumbency as for active (persistent or recurrent) tuberculosis of the spine and await results, whether such disease is clinically present or not. With the passage of time Type III paraplegia is then found to fall into one or other of two subdivisions :—

Type IIIa.—Late onset paraplegia recovering on conservative treatment as for active tuberculosis of the spine.

Type IIIb.—Late onset paraplegia not so recovering.

Recovery in Type IIIa usually commences under treatment within nine months. The writer has, however, seen late onset paraplegia remain stationary under treatment for eighteen months and then recover completely (E. C., age 13, Pyrford Orthopædic Hospital, 1931).

The direct mortality rate from late onset paraplegia is negligible. Skin sores do not occur and ascending urinary infection is rare.

PROTEIN OF THE CEREBRO-SPINAL FLUID IN LATE ONSET PARAPLEGIA IN RELATION TO END RESULTS OF TREATMENT.

SERIAL NUMBER OF PATIENT	PERSISTENCE OF PARAPLEGIA PRIOR TO ESTIMATION	PROTEIN IN CEREBROSPINAL FLUID*	RESULT OF FURTHER CONSERVATIVE TREATMENT OF THE PARAPLEGIA AS FOR ACTIVE TUBERCULOUS DISEASE OF THE SPINE	
	Months	G. per 100 c.c.		
(S) 58 ..	4	0·9	Recovered	—
(B) W.N. ..	4	0·4	Recovered	—
(B) D.S. ..	1	0·3	Recovered	—
(S) 68 ..	1	0·24	Recovered	—
(B) E.C. ..	3	0·15	Recovered	—
(B) J.B. ..	12	0·1	Recovered	—
(S) 99 ..	10	0·1	—	No Recovery
(S) 22 ..	2	0·08	Recovered	—
(B) J.D. ..	10	0·06	—	No Recovery
(B) E.B. ..	6	0·05	—	No Recovery
(S) 97 ..	2	0·03	—	No Recovery
(S) 32 ..	24	0·015	—	No Recovery

* Normal 0·01–0·03

THE PATHOLOGY AND ETIOLOGY OF TYPE III PARAPLEGIA

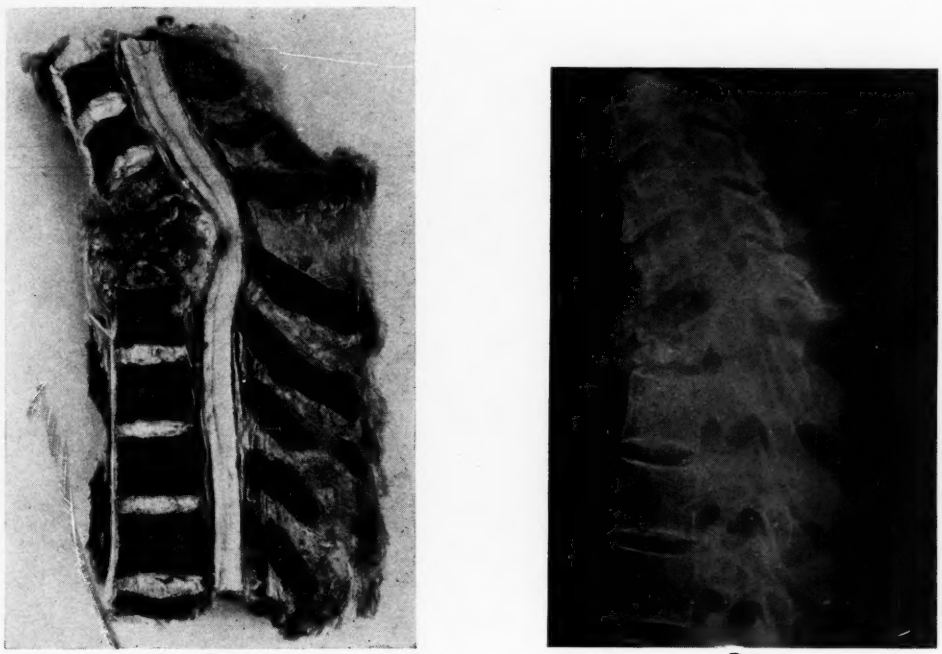
Inquiry into the pathology of Type III paraplegia is hampered by the difficulty of obtaining specimens of the spine from such patients, since they seldom die in hospital. Certain specimens are, however, available which prove, as we have suspected on clinical grounds, that the cause of the paralysis may be activity of the old tuberculous disease in the spine. It is desirable to emphasize that such activity is in whole or part of the old bone lesion where it abuts upon the cord. It is no new process such as a tuberculous pachymeningitis, a term often used in the past to explain away any form of paraplegia in Pott's disease to which no definite origin could be assigned.

Case (B) W. N., Addenbrooke's Hospital, Cambridge. A man, aged 52, was seen in June, 1933, with a late onset paraplegia of five months' duration. Both legs were markedly spastic, but there was some voluntary power in all muscle groups except the dorsiflexors of the toes and feet. There was patchy anæsthesia up to the 2nd thoracic segment, most complete below the knees, and partial urinary incontinence. He had a marked kyphos at the cervico-thoracic junction, which had been present unchanged for twelve years. When this

kyphos had first appeared he had had a good deal of pain, both root pain and locally at the site of disease. He had never received any treatment for this, having managed to keep on at his work as a farm labourer, and for ten years since had had no symptoms. At the time of examination he had also an early active focus of tuberculosis in the low lumbar spine, but clearly this was in no way associated with the paralysis. Clinically and by X-ray the old cervico-thoracic lesion appeared in all probability quiescent.

He was rested in recumbency, and for sixteen weeks showed steady improvement. Voluntary power returned to all muscle groups in the legs, and anæsthesia was present only below the knees and in one area on the abdominal wall. Bladder control became normal. The upper abdominal reflexes, previously absent, reappeared, but the lower were still absent.

On Oct. 27, 1933, he had a thrombosis of the right iliac vein following pressure by a retroperitoneal abscess arising in connection with the lumbar disease. He died the next day after pulmonary embolism.



A B

FIG. 535.—*Case* (B) W.N. A, Specimen of spine from a patient with late onset paraplegia Type IIIa. Active disease is present in a chronic form. The cord is compressed by a mass of granulation tissue and caseous material containing many areas of calcification. B, X-ray of specimen. (*Author's specimen, Addenbrooke's Hospital, Cambridge.*)

Examination of the spine of this patient (*Fig.* 535) showed re-activity of the old cervico-thoracic lesion, with many of the main features which might be expected in paraplegia of Type I with early active disease. Tuberculous granulation tissue and caseous material occupied the extradural space, the cord being compressed thereby against the laminæ. At the site of the main bone destruction and in front of this was an abscess containing caseous material.

The most remarkable feature of this case seems to be that in spite of such gross changes the paraplegia was recovering steadily, and, indeed, for a Type III paraplegia, rather rapidly. In contrast, however, with the appearances in early active disease the granulation tissue and caseous matter were semi-calcified. Moreover, the surrounding vascular engorgement and œdema so characteristic of early disease were almost entirely absent.

On microscopy the dura mater was found to be forming an efficient barrier to the spread of tuberculosis and was marked off sharply from the granulation tissue without, but in its outer layers showed rather more round-celled infiltration than is usual in similar sections with early active disease. Sections of the cord showed round-celled infiltration about the vessels, and some degree of vacuolation in the white matter. The appearances were hard to interpret exactly, but on the

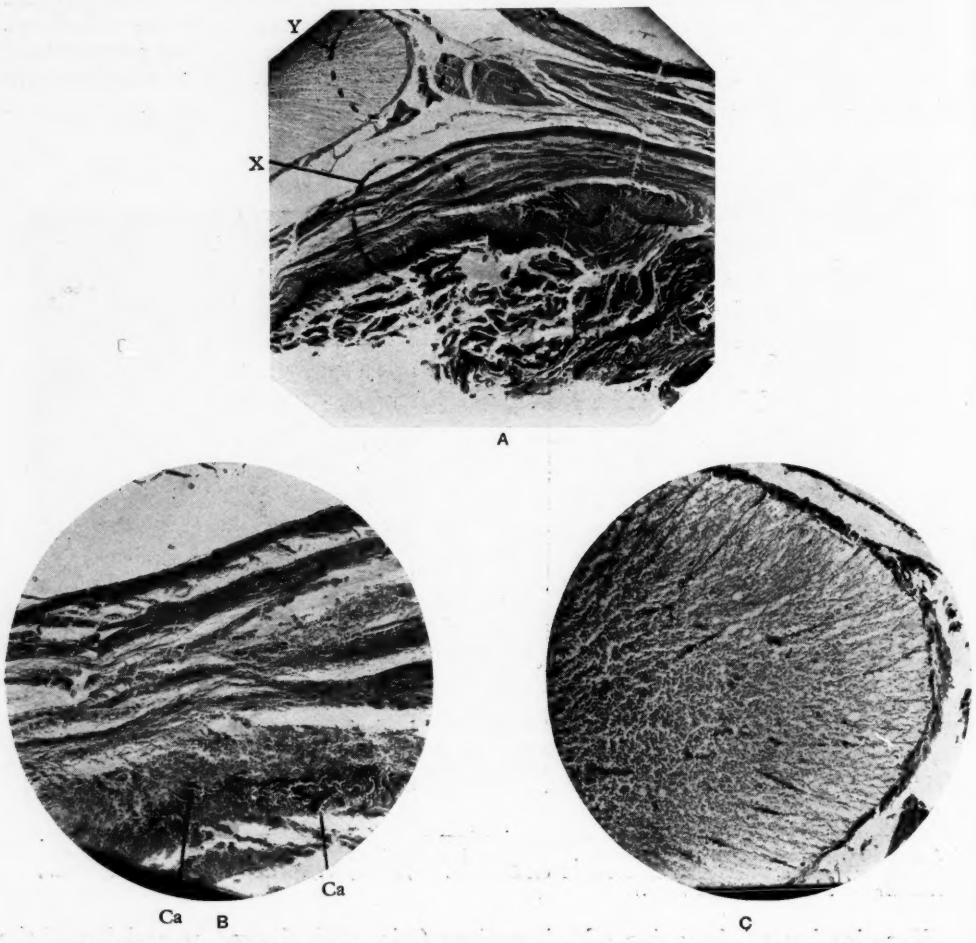


FIG. 536.—*Case* (B) W.N. Sections from same specimen as *Fig.* 535. A, Section showing both active and calcified tuberculous tissue lying against the intact dura mater. (X) Area shown in B. (Y) Area shown in C. B, Section showing, above, the thickened and sclerotic but intact dura mater, and below tuberculous tissue containing active areas with well-marked giant-cell systems and many deposits of calcium (Ca). C, Part of the cord showing considerable atrophy of nerve-cells and fibres and perivascular cellular infiltration and fibrosis.

whole there would appear to be less vascular engorgement and vacuolation both in the grey and white matter, and more permanent atrophy with disappearance of nerve-cells and fibres, than in paraplegia of Type I (*Fig.* 536).

The clinical differences between this paraplegia and that of paraplegia of Type I must in fact have been dependent upon the relative inactivity of this chronic tuberculous focus as opposed to a similar focus at an early active stage. *This chronicity, with the consequent lack of surrounding toxic and vascular change,*

seems to be the essential point to be appreciated in this case. The actual mechanical pressure upon the cord had no doubt arisen very slowly, and, as Gowers[15] has pointed out, the effect of pressure upon the cord is directly proportionate to the rapidity with which it comes about.

Another specimen of great interest is one preserved in the Pathology Museum of the Medical School at University College Hospital, which shows the condition of the spine and spinal cord in a girl who died from intercurrent disease following recovery several years earlier from a late onset paraplegia (*Specimen No.* 16 *B.S.*). The specimen (*Fig.* 537) is from a girl treated many years ago at University College Hospital. She was first seen with a thoracic kyphos at the age of 6, but as the disease was not considered active was allowed to remain at home. In the next three

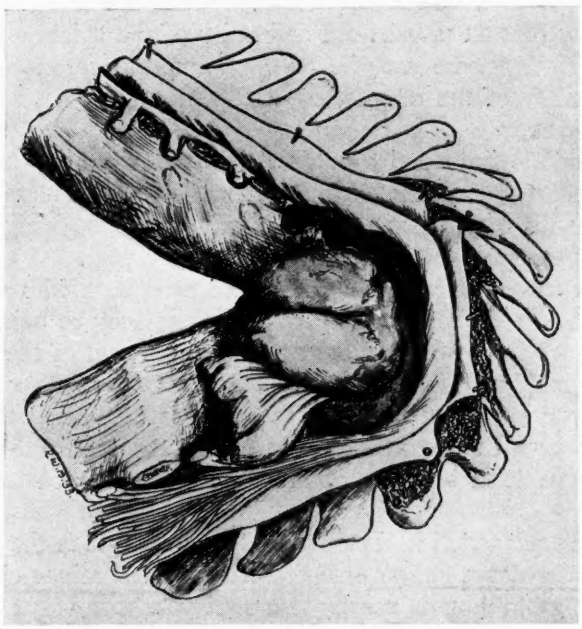


FIG. 537.—Specimen showing the spine of a girl who died from intercurrent disease six years after recovery from a late onset paraplegia of Type IIIa. There is no mechanical constriction of the cord. Anteriorly the cord is lying against masses of calcified débris and old tuberculous material, and in front and on both sides of this are large paravertebral abscesses. Case report and description in text (and in *Illustrated Medical News*, 1889, cxciii).

or four years the deformity increased and plaster jackets were applied. In the light of our present knowledge of thoracic Pott's disease in a child there is no doubt that the disease was active during these years. Six years after being first seen—at the age of 12—she developed a widespread late onset paraplegia from which, however, she recovered on treatment in hospital. For a further six years she remained in good health. At the age of 18 she died of bronchitis.

It is clear from the specimen that even at the time of death there was no assurance that the tuberculosis of the spine was permanently healed. Big semi-calcified paravertebral abscesses and masses of débris lie in the concavity of the kyphos in close relation to the cord. These form a site in which live organisms might lie dormant for years, finally becoming active again and being once more

the cause of reactionary changes in the cord. In this specimen there is no evidence that there had ever been true compression of the cord, which is of apparently normal calibre and has accommodated itself well to changes in the vertebral canal, though the latter is angulated at two points by more than sixty degrees. If any compression was present at the time of the paralysis, it has disappeared spontaneously. There seems to be no alternative to the suggestion that the late onset paraplegia in this girl was associated with persistent or renewed activity of the spinal lesion.

When we consider the clinical findings in conjunction with such specimens as the above it seems certain that the vast majority at least of late onset paraplegias which recover under treatment (Type IIIa) are due to such persistence or reactivity of the spinal disease, and since Type IIIa and Type IIIb paraplegias are usually indistinguishable at their onset, the relevance of this with regard to treatment of late onset paraplegia as a whole is obvious.

It seems likely that an unbalanced deformity of the spine at the end of treatment for the original disease may be of great importance in keeping up or in reprecipitating activity of the disease, especially in a child, in whom bone fusion may be long delayed.

The weight of the head and shoulders falling in front of the old focus must exert upon it a constant levering action and must undoubtedly tend to maintain a state of strain, with reaction in the bones and soft parts, which can but favour further activity of the disease (*see* Seddon's article, p. 793).

What now of those patients with late onset paraplegia who fail to recover on conservative treatment—Type IIIb? Clinically there may be evidence that disease of the spine is active in these patients at the time of onset of the paralysis, just as there is at times in Type IIIa. It is probable that much Type IIIb paralysis is at its onset of Type IIIa, and that just as in Type I some factor may be added which makes recovery of the cord impossible and turns it into Type II, so in these patients cord degeneration becomes established for one reason or another as time goes on.

The simplest explanation is that Type IIIb paraplegia is the result of a slowly progressive avascular degeneration arising from repeated minor circulatory damage by chronic disease grumbling on nearby year after year with very little outward sign.

How far pressure upon the cord by bone or hard débris is sometimes the primary cause of late onset paraplegia is hard to assess. Sometimes it may be a primary factor. At any rate, true compression by bone has been found at operation and after death in late onset paraplegia, and Seddon describes such cases, but such compression would not have arisen without reactive disease which allowed of further bone destruction, and it is the occurrence of this reactive disease which requires emphasis. As mentioned when discussing Type II paraplegia, gross primary compression by bone is probably always associated with rapid destruction, and may be expected to occur therefore almost entirely with early disease. Frequently at post-mortem examination of a patient with irrecoverable late onset paraplegia the cord is found stretched over a mass or bar of bone in the anterior wall of the canal, but it must be remembered that once some degree of cord atrophy is established the cord will lose in length and elasticity, and will therefore be drawn forward to lie against the anterior wall of the canal, which, at the best, will consist of rough calcified débris and scar tissue and may perhaps be still the site of a low-grade infection. Further embarrassment of the cord by mechanical irritation will

then occur and a vicious circle will be established, still further cord degeneration following.

With late onset paraplegia there is sometimes a history of a recent increase in the kyphos. With most this is doubtless a sign of active disease with some further destruction of bone: with some, especially with children in whom growth is occurring, it may at times be simply the result of faulty mechanics in an unbalanced spine. In any case, an increasing kyphos will add to the possibility of the pushing back of débris towards the cord, and this, even if it produces no gross compression, will add to the likelihood of an irritation factor being concerned in the production of cord degeneration.

The following case of Type IIIb paraplegia provides some interesting evidence in respect to the above possibilities.

Case (B) E.S.81.—A girl, aged 3, was admitted to Queen Mary's Hospital, Carshalton, under Dr. Pugh, with early mid-thoracic Pott's disease, and underwent two years' conservative treatment. Thereafter she was at home for four years in perfect health. At the age of 9 she developed a partial, but steadily deepening, paraplegia. She was readmitted to hospital with an incomplete motor and sensory paraplegia with double incontinence.

Under conservative treatment on a frame she made considerable progress at first, but later the paralysis again deepened and became stationary. After three years, being now aged 12, she had very little voluntary power in the lower limbs except in the dorsiflexors and plantar flexors of the feet and toes.

Sensory loss was patchy to the mid thighs on both sides, and sphincter control was imperfect. It was decided to perform a laminectomy in the hope of relieving the cord from some mechanical pressure. At operation the cord was found attenuated and flattened lying against the front wall of the canal. Posteriorly there was plenty of room in the extradural space and there was no evidence of narrowing of the spinal canal as a whole. The girl died of post-operative shock.

Examination of the specimen of the spine and cord (*Fig.* 538) shows that the disease had evidently become quiescent in the last years before death. At first sight there would appear to be compression of the cord by a bony mass pushed back from the main area of disease where the remains of three vertebral bodies are fused into a solid mass.

The bony canal has been much shortened by the angulation so that, unless the cord were even more shortened so that it is compelled to take the shortest possible course against the anterior wall of the canal, it would be lying in the posterior part of the canal where there is room to accommodate it. The relationship of the bony walls of the canal to the cord are not easy to make out in the drawing of the specimen, as destruction of the laminæ at operation obscures the true relationships, but the accompanying diagram makes this clear (*Fig.* 538 B). The dura mater is quite unattached to the wall of the canal anteriorly. Microscopy of the cord (*Fig.* 539) shows general atrophy, with complete loss of differentiation of grey and white matter—though a fair number of medullated fibres are still present. Blood-vessels in the cord itself and in the meninges are scanty and show perivascular fibrosis. The subarachnoid space is crossed by a few fine adhesions. The dura mater is somewhat thickened, especially antero-laterally, and both in the dura mater and in the region without, which was probably at one time invaded by tuberculous granulation tissue, there are deposits of calcium. This specimen is interpreted, therefore, as showing primary cord atrophy rather than atrophy secondary to compression by bone.

A

B

C

FIG. 538.—*Case* (B) E.S. 81. A, Specimen from a girl aged 12 with an irrecoverable late onset paraplegia Type IIIb. Laminectomy has been performed. The atrophic and shrunken cord is seen lying against the fused mass of bone forming the anterior wall of the canal. B, Diagram to show relationship of walls of vertebral canal to the cord. C, X-ray taken shortly before death. The specimen is at Queen Mary's Hospital, Carshalton.

In no specimens of late onset paraplegia, any more than in paraplegia with early active disease, has the writer seen any evidence of chronic tuberculous pachymeningitis as the cause of the paralysis.

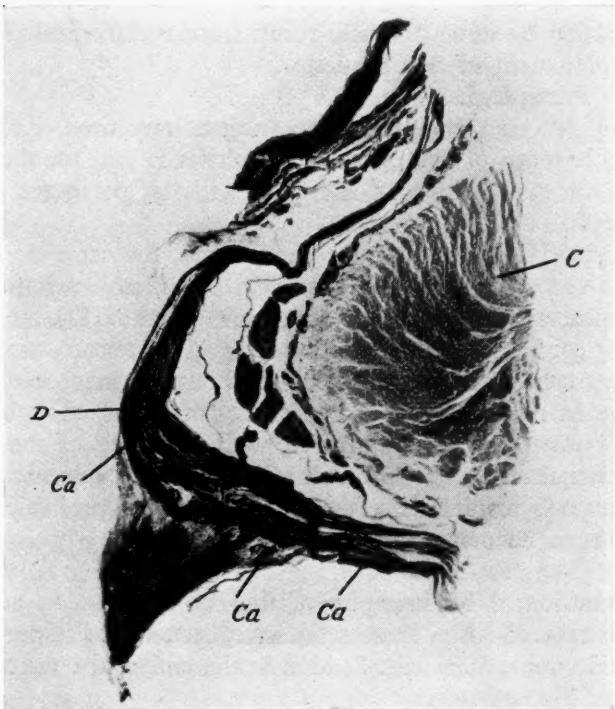


FIG. 539.—*Case* (B) E.S. 81. Half section of the cord at the level of the old disease from the specimen shown in *Fig.* 538. *C*, Atrophic cord without differentiation between white and grey matter. A fair number of intact axons are however present. Very few nerve-cells are visible. *D*, Somewhat thickened dura mater showing many deposits of calcium (*Ca*).

SUMMARY

A survey has been made of a series of 186 patients with paraplegia in Pott's disease.

From a clinical study of these and from an investigation of the post-mortem pathology it is suggested that paraplegia in Pott's disease falls into certain clinical types with which are associated constant etiological factors.

Early Onset Paraplegia.—

Type I.—Paraplegia with early active disease, arising, persisting, and diminishing in direct relationship to the activity of the tuberculous infection of the spine. The only constant etiological factor is the presence of active tuberculosis in immediate relationship to the dura mater. In all such the paralysis is associated with much toxic and vascular reaction in the cord, and in some this alone is the cause of the paraplegia. The importance of this reaction in all paralyses where active disease is present is emphasized. In many the additional factor of true compression of the cord by granulation tissue or pus is also present.

Type II.—Paraplegia associated in onset with early active disease but persisting

on permanently even if tuberculous infection in the spine has become completely quiescent. The causative factors in the early stages are usually the same as in Type I. In the permanent stage there is in many simply a varying degree of cord atrophy from reaction to too long continued activity of the disease before quiescence is attained. In others there is true compression of the cord by bone or débris. Compression by bone can only result from pathological dislocation of the spine or the displacement of free sequestra.

Late Onset Paraplegia.—

Type III.—Late onset paraplegia appearing at any time, up to many years, after apparent quiescence of the disease. Emphasis is laid on the fact that most such paraplegias are associated with low-grade activity, persistent or recurrent, of the spinal infection.

In some (Type IIIa) the causative factors are the same as in Type I, but modified, just as the disease itself is modified. In many such the paralysis will recover with conservative treatment. In others (Type IIIb) the main causative factor is avascular cord degeneration from too long persistent reaction to the disease.

True primary cord compression by bone, though it may occur, has comparatively small place in the causation of late onset paraplegia, though apparent compression, which is due to primary atrophy, with shrinking of the cord so that it comes to lie later against the anterior wall of the canal, is common.

Tuberculous pachymeningitis, often mentioned as a probable cause of paraplegia, has not been encountered.

To save repetition, a bibliography of the subject, and an acknowledgement of the writer's indebtedness to those who have been of assistance to him in this work, are given in conjunction with Seddon at the end of the next paper.

POTT'S PARAPLEGIA: PROGNOSIS AND TREATMENT

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PART I. THE CLINICAL CLASSIFICATION OF CASES OF POTT'S PARAPLEGIA IN RELATION TO PROGNOSIS

In any disease the practical value of a clinical-pathological classification is proportionate to the reliability of the guidance it gives in regard both to prognosis and treatment; and this is particularly true of Pott's paraplegia.

The only serious attempt hitherto to deal with Pott's paraplegia along these lines has been made by Mme Sorrel-Dejerine, whose weighty monograph[30] has won acceptance in many places and raised storms of opposition in others. It is sufficient to say that Butler and I have been quite unable to fit even the majority of our 186 cases into the attractive "early-abscess-recovery" and "late-pachymeningitis-permanent paralysis" groups which she described—after the study of only 40 cases. Not only did some of our early cases fail to recover, and many of our late cases recover completely, but in the specimens from cases of late paraplegia that we were able to examine there was no evidence whatever of pachymeningitis, the condition which Sorrel-Dejerine holds responsible for most of the late cases of paraplegia. I regret that considerations of space prevent me from appraising her thesis in detail, but the question has already been referred to by Butler and we hope to deal with the matter more fully elsewhere at a later date.

The first part of this paper is an attempt to show that the classification of the various types of Pott's paraplegia agreed on by Butler and myself is a fairly accurate guide to the prognosis in this disease.

It is generally agreed that with good conservative treatment, 60 to 90 per cent of all cases of Pott's paraplegia recover completely. In my series of 100 cases there were 109 attacks of paraplegia. Seven histories are too recent to be of statistical value: in the remaining 102, there were 71 complete and 3 partial recoveries, and 28 patients were permanently paralysed. This gives a recovery rate of about 70 per cent. There were 21 deaths, of which 8 were directly attributable to the paraplegia. There is no doubt that the death-rate in spinal caries is considerably increased by the incidence of paraplegia.[38]

Must we accept this proportion of permanent or fatal paraplegias as inevitable, and if so, is there any way of recognizing the unfavourable cases in advance? Before attempting to answer this question it is necessary to consider certain clinical signs that are of importance in prognosis, quite apart from the clinical-pathological types to be described later. These signs are common to all forms of Pott's paraplegia.

Age.—I am in entire agreement with Sorrel-Dejerine in finding that recovery from paraplegia is not greatly influenced by the age of the patient. In my series the chance of recovery improved slightly as the age increased up to 60 years, but

the two patients who developed paraplegia after this age died within eighteen months without recovering from their paralysis.

Paraplegia in Extension.—This indicates an incomplete lesion of the cord—that is to say, certain tracts have escaped compression; but although the prognosis in this condition is good, it does not necessarily follow that the damage done is always reparable. Out of 67 patients with paraplegia in extension, 54 recovered completely, 3 recovered incompletely, 2 recovered after several years, and 8 (patients in whom paraplegia in extension changed later to paraplegia in flexion) were permanently paralysed. Painful and excessive spasm of the limbs in paraplegia in extension is not an unfavourable sign.

Paraplegia in Flexion.—In paraplegia in flexion, which indicates involvement of the whole thickness of the cord, the prognosis is bad. All 12 cases with this condition were permanently paralysed. However, a case of recovery after paraplegia in flexion has been recorded by Souques[33] in a woman aged 51 years, and one of Butler's 15 cases of paraplegia in flexion recovered almost completely.

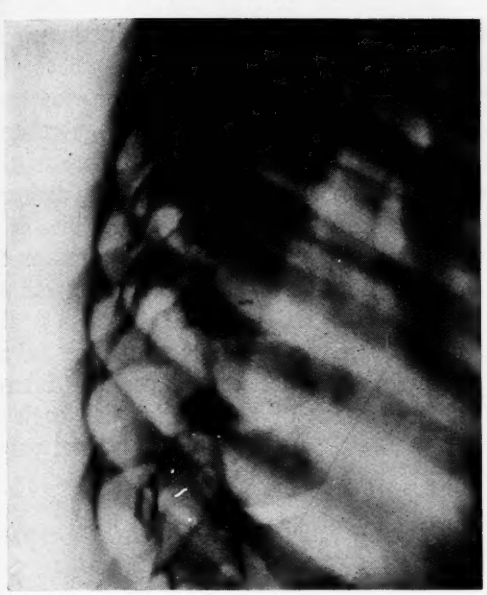
Flaccid Paralysis.—In this the prognosis is equally bad. Although spasticity may return, complete recovery is almost unknown. In 1 case out of 7 the patient recovered a little voluntary power in the lower limbs a few months before he died of amyloid disease. The other 6 patients were permanently paralysed.

Duration of Motor Paralysis.—In the past considerable attention has been paid to the duration of paralysis, and as motor changes generally appear long before, and remain for some time after, sensory changes are present, it is the duration of *motor* paralysis that has generally been studied. I have not found my own or the published statistics of the total duration of paralysis of the slightest value in prognosis. As recovery may sometimes occur after paraplegia lasting for as long as six (*Case* S.5) or seven (*Case* S.7) years, a prognosis based on the length of time that paraplegia has been present resolves itself into nothing more than a pious hope that recovery will occur in any case of paraplegia in extension of not more than seven or eight years' duration.

A far more exact prognosis may be made from a study of the duration of total motor paralysis. This must not be confused with complete paraplegia, i.e., paraplegia in flexion. By total motor paralysis is meant paraplegia in extension with complete loss of voluntary power: we already know that complete loss of voluntary power resulting from paraplegia in flexion or flaccid paralysis carries a hopeless prognosis. If voluntary power is never entirely lost, recovery is assured. In my series there were 21 cases in which some degree of voluntary power was always present; every one made a complete recovery, though in one case (*Case* S.7) it was delayed for seven years. On the other hand, where total motor paralysis is present, the prognosis becomes rapidly worse with each month that elapses after its onset; there were 13 recoveries after three months of total motor paralysis, 11 after six months, 3 complete and 1 incomplete recovery after nine months, 2 after one year, 4 complete and 3 very incomplete recoveries in the second and subsequent years. *The chances of complete recovery from total motor paralysis of more than six months' duration are extremely poor.*

Sensory Changes.—A study of the sensory changes is of little value in prognosis. It may be said that as a rule the prognosis is good in cases where sensory changes are absent, but the return of sensation in a case where it has been lost, though encouraging, does not necessarily indicate that complete recovery can be expected.

A number of cases in this series recovered sensation completely and yet the motor paralysis was permanent. Sorrel-Dejerine believes that the prognosis is bad in



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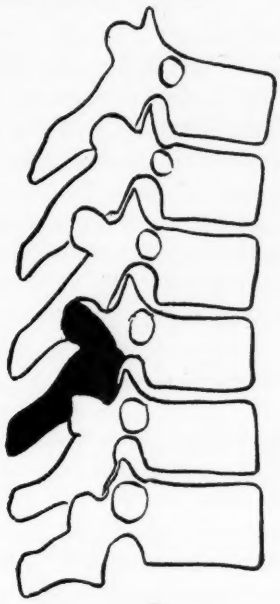
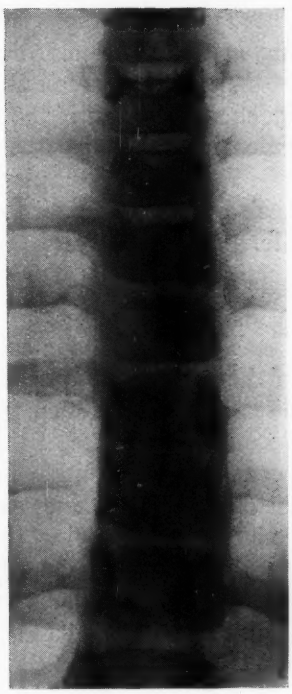


B

FIG. 540.—*Case* S.36. A, Lateral X-ray of the spine of a boy of 9 who was admitted to hospital with mild paraplegia. There is a destructive lesion of the pedicles, laminæ, articular processes, and spinous process of Th.8. A typical lesion of the vertebral bodies appeared later; the paraplegia cleared up spontaneously. B, Explanatory drawing of A. C, X-ray, showing lesion at A. (*Dr. J. G. Johnstone's case. Princess Mary's Hospital, London County Council, Margate.*)



A ····

C

cases where sensory changes make their appearance many months after the onset of a purely motor paraplegia; this was true in four of my cases, but not in the remaining two.

There is, however, one form of sensation the absence of which is of great prognostic significance. Vibration sense is lost only in cases where compression is so severe that the posterior columns of the cord are involved. When the vibrations of a tuning-fork cannot be felt on the bony points in the lower limbs the prognosis is bad. Recovery from paralysis occurred in only 2 out of 11 patients who had lost vibration sense in the lower limbs. One of the patients who recovered (*Case* S.36) is of unusual interest: this child suffered from paraplegia as a result of disease involving a thoracic neural arch (*Fig.* 540), and loss of vibration sense was present even when the motor paralysis was well on the way to recovery. Presumably the lesion producing the paraplegia affected the cord from behind forwards

instead of in the usual way, and derangement of conduction in the posterior columns persisted when the pyramidal tracts had almost recovered.

Sphincter Changes.—The absence of derangement of function in the bladder and rectum is an encouraging sign. Only 4 of the 33 patients who remained entirely free from sphincter trouble were permanently paralysed. In cases where the bladder and rectum are involved the recovery of normal sphincter action does not always indicate that the paraplegia will clear up completely; in *Case* S.19 control returned in six months, and yet this child is permanently paralysed. One of the causes of death in patients with involvement of the bladder is ascending urinary infection, yet only a few fatalities can be attributed to this complication, and it is remarkable how readily severe infection of the urinary tract responds to treatment, even though the paraplegia is permanent. Severe urinary infection occurred in 6 of my cases, but in none was it the cause of death.

Pregnancy.—According to Myers[22] pregnancy may determine the onset of an attack of paraplegia in a patient with incompletely healed spinal caries. I have seen one patient with spinal caries who had an attack of paraplegia with each of four pregnancies, and in one of Butler's cases (*Case* B.7) the onset of a fatal paraplegia coincided with pregnancy.

THE CLINICAL-PATHOLOGICAL TYPES

The classification proposed by Butler distinguishes three types of Pott's paraplegia:—

Type I.—Paraplegia with early active disease; arising, persisting, and diminishing in direct relationship to the activity of the tuberculous lesion in the spine.

Type II.—Paraplegia associated in onset with early active disease, but persisting indefinitely—even if the tuberculous infection in the spine has become completely quiescent.

Type III.—Paraplegia occurring after a tuberculous spine appears to have healed; often in patients who have enjoyed many years of good health and active life.

TYPE I PARAPLEGIA

Type I has been so familiar since the time of Pott that a detailed description of the condition is no longer necessary. Paraplegia may be the first sign of spinal disease, and in adults, particularly, often occurs before any deformity is apparent. Secondly, weakness of the lower limbs may be complained of by a patient who has been aware of trouble in the back for some weeks or months; in this case the history is "back first—then legs". Lastly, paraplegia may develop in cases of active caries receiving the most efficient hospital treatment.

It has been shown by many observers, and again by Butler and myself, that this type of paraplegia is the result of the presence of active tuberculous disease in the immediate vicinity of the cord—the 'abscess' of former writers (*see* Butler's article, p. 744)—and the waxing and waning of activity in this tuberculous focus will determine the course of the paraplegia. Usually the paraplegia is mild at first, then severe—often with sensory disturbances and sphincter involvement; finally, after several months, signs of improvement appear, and at last after a painful and anxious ordeal, the patient recovers full control of his lower

limbs. In a few cases the course of the paralysis is less severe, and never amounts to more than a few weeks or months of spasticity and weakness of the lower limbs.

Every case conforming at first to this type will recover unless certain things happen :—

1. The initial infection may be so virulent that the patient dies rapidly of tuberculosis before the paraplegia has run its course. We are all familiar with these tragic cases of overwhelming tuberculous disease in which the spinal lesion is merely incidental; the most that can be said of the paraplegia is that it hastens the end.

2. The disease persists for so long before finally healing that the damage to the cord, reparable at first, ultimately becomes permanent. Activity has persisted for longer than the cord can tolerate.

3. An 'accident' occurs, vascular or skeletal, resulting in permanent damage to the cord.

TYPE II PARAPLEGIA

Type II comprises the second and third contingencies just described. The Type II case is therefore generally a Type I case 'gone wrong'; however, it is sometimes a Type II from the first, without any chance of recovery from the paralysis even in the earliest days. Fortunately, it is often possible to recognize the Type II case in advance, which is of value in prognosis and treatment; there are certain danger signals that herald the Type II paraplegia, and they must constantly be watched for.

1. Paraplegia of Sudden Onset Rapidly Becoming Complete.

Two conditions are known to be responsible for this syndrome, and in both the prognosis is bad.

'Concertina' Collapse of a Vertebral Body.—A good example of this condition is described by Girdlestone[13] (Case 4), which is typical of those recorded by other observers.

A patient, aged 21 years, was admitted to hospital with backache of two weeks', and paralysis of the legs of six weeks', duration. On admission there was complete flaccid paralysis of the lower limbs, loss of all forms of sensation below the pelvis, and double incontinence; X-ray examination showed collapse of the body of Th.11 with almost normal intervertebral discs above and below. Laminectomy was performed immediately; "after full exposure, flattened area opposite D.11 filled out, but pulsation never seemed as full as at higher level. There was a prominence of the theca opposite D.11. An aneurysm needle passed in front of theca at this level failed to find débris."

The operation was not followed by any immediate improvement. Some degree of recovery occurred ultimately, but spasticity of the lower limbs and incomplete sphincter control persisted.

The cardinal signs of concertina collapse are total paraplegia of early and rapid onset, and the radiographic demonstration of collapse of a tuberculous vertebral body.

A case of Butler's is of unusual interest in that paraplegia due to concertina collapse was superimposed on a Type I paraplegia.

A boy of 19 was admitted in November, 1932, to the Shropshire Orthopædic Hospital, Oswestry, under the care of Mr. Harry Platt. There was a history of three months' weakness in the lower limbs. On admission the patient was found to have a Type I paraplegia due to a lesion at the thoraco-lumbar junction.

Early March, 1933.—X-ray showed narrowing of the space between Th.8 and 9. ? second focus of disease.

Late March, 1933.—Sudden deepening of paraplegia; level of sensory disturbance rose to Th.8, voluntary power entirely absent, loss of sphincter control. X rays showed partial collapse of Th.8 with destruction of pedicles and transverse processes.

FIG. 541.—Concertina collapse of the eighth thoracic vertebral body. Diagnosis confirmed histologically. (*Reproduced by kind permission of Mr. R. W. Butler.*)

April 22, 1933.—Laminectomy by Mr. Platt. The laminæ and overlying muscles were found to be infiltrated with material suggesting a new growth. Operation abandoned. Histological examination, however, confirmed the diagnosis of tuberculosis. Further operation not undertaken on account of widespread local disease.

Sept. 22, 1933.—Patient died. At autopsy: tuberculous lungs, mesenteric glands, and right kidney. Diagnosis of concertina collapse of Th.8 confirmed.

It is impossible to separate the effect on the cord of the lesion in the body of Th.8 from that due to the disease in the laminæ and pedicles, but in view of the sudden deepening of the paralysis it is reasonable to suppose that the compression of the cord at the higher level was due to the rapid collapse of the body of Th.8 (*Fig.* 541).

Acute Thrombosis of Vessels Supplying the Cord.—The history in this condition is very similar and the prognosis is hopeless.

Case S.84.—A boy, aged 18 years, admitted to St. Luke's Hospital (London County Council), Lowestoft, under the care of Dr. H. J. A. Colvin, with caries of C.4–6. Enormous abscess in the left neck. Two months after he first came under observation complete paralysis of all four limbs suddenly developed, the condition resembling the 'spinal shock' of traumatic lesions. Death within three weeks: paralysis flaccid to the last. At autopsy the large abscess was found to extend between the bodies of C.4 and 5 on to the anterior surface of the dura, but it is doubtful if it exerted any effective pressure on the cord, which was of normal contour; there was no obvious anatomical cause for the paraplegia. On opening the large abscess cavity to the left of the vertebral column, it was found that the wall of the abscess lay in intimate contact with the left vertebral artery, which contained a mural clot: the right artery was normal. Horizontal serial sections show that the clot had spread into the spinal branch of the artery that runs along the nerve-root and enters the cord at the level of the lesion (*Fig.* 542); other smaller vessels around the nerve-root are thrombosed. Sections of the cord itself show that although the superficial vessels appear normal, there is widespread thrombosis of the vessels in the substance of the cord (*Fig.* 543). The white matter is degenerate, though the grey matter, particularly in the anterior horns, appears normal; the central canal is patent. There is only slight dilatation of the vessels below the lesion.

The striking features of this condition—sudden complete paralysis, but nothing apart from the usual tuberculous lesion radiographically—are characteristic of a serious vascular lesion of the cord. Elsberg,[8] though citing no evidence to prove his point, believes that this type of paraplegia is the result of a vascular lesion, and that operative interference is contra-indicated.

For the differentiation of these two serious accidents we are dependent upon X-ray examination. In concertina collapse the crushing of the vertebral body is easily identified, though it may be difficult to distinguish from the collapsed vertebra of secondary carcinoma: in acute thrombosis the tuberculous focus is of the usual type.

FIG. 542.—*Case* S.84. Infective thrombosis of the vertebral artery spreading along a spinal branch and involving most of the small vessels of the corresponding segment of the cord. Horizontal section through the contents of the intervertebral foramen and the vertebral artery.

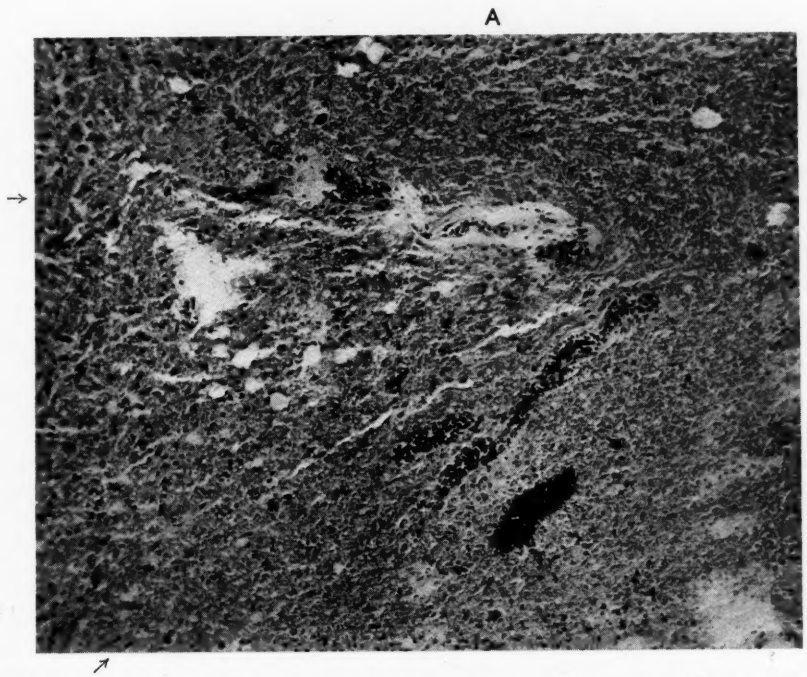


FIG. 543.—*Case* S.84. Two small vessels in the grey matter of the cord cut obliquely. The lumen is completely obliterated in each case by clot densely infiltrated with leucocytes. The wall of the upper vessel is thickened at A. The cellular infiltration of the area of grey matter shown is marked enough to constitute a localized myelitis. Most of the changes, however, were confined to the white matter.

2. Spinal Tumour Syndrome

This puzzling condition (*Fig.* 544), fortunately rare, is likely to be encountered by the neurologist rather than the orthopædic surgeon, seeing that apart from operation it is indistinguishable from true spinal tumour. It is the only form of Pott's paraplegia in which neurological signs develop before radiographic changes are demonstrable in the spine. *Case* S.67 is a good example of this condition.

Fig. 544.—From a woman aged 23 years, who died with paraplegia. Although there is widespread infection of the bodies of the 4th and 5th cervical vertebræ as evidenced by the loss of normal trabeculation in the spongy tissue, and some erosion of the posterior surfaces of both bodies, nothing abnormal could be found in the X-ray. The abscess responsible for the compression is well shown. (*Guy's Hospital Museum.*)

Case S.67.—F. G., a man 37 years of age, was admitted to the Westminster Hospital on June 7, 1926, under the care of Dr. Hildred Carlill. There was a history of six months' backache, steadily becoming more severe, and increasing weakness in the lower limbs of two months' duration. On admission, paraplegia in extension with no voluntary power: cutaneous analgesia below Th.7. Cisternal C.S.F. normal; lumbar C.S.F. yellow, cells less than 1 per c.mm., protein 0·35 per cent, globulin present, gold curve 000001223, W.R. negative. X-ray showed no bony lesion in spine; lipiodol completely arrested at level of Th.9; subarachnoid space displaced to right. Diagnosis, extra-medullary extra-thecal tumour.

June 14, 1926.—Laminectomy by Mr. Rock Carling: Th.9–12. Cord found displaced to right at level of Th.10, pulsation good above, poor below. During examination of theca, pus emerged from a mass of granulation tissue at the level of Th.12: track found passing upwards for 1½ in. on left of cord, i.e., to level of Th.10. Track irrigated through small catheter; wound closed. Material removed positive for T.B. Paraplegia cleared up completely within eight weeks, but the laminectomy wound never healed and discharged large quantities of pus. Patient died in October, 1927, probably from amyloid disease. Before death, lesions involving the bodies of Th.7–8, 10–11 were demonstrated radiographically (at St. Luke's Hospital, London County Council, Lowestoft).

In another case, S.80, a similar lesion was found in a man 21 years of age. However, in spite of early laminectomy, the paralysis was permanent, though the patient survived.

The spinal tumour type of Pott's paraplegia is a peculiarly lethal condition. The cases recorded by Fischer[11] and Vincent and Darquier[39] terminated fatally. I believe that *Case* S.67 is the only one described in which recovery from the paraplegia occurred before death, and this was undoubtedly attributable to the prompt operative intervention. The spinal tumour syndrome is therefore very definitely a Type II condition: recovery from paralysis has never been known to occur apart from operation.

Minimal Bone Destruction.—It occasionally happens that paraplegia appears in cases where the amount of bone destroyed by the disease is extremely small: there were three such cases in my series. In *Case* S.14 the paraplegia was permanent; in *Case* S.5 the paraplegia took four years to clear up, then relapsed and persisted for a further six years; in *Case* S.7 (*Fig.* 545) paraplegia was present for seven years. The association of slight bone destruction with permanent or long-standing paralysis seems to be more than a coincidence. Can it be that on account of the small amount of bone destroyed the vertebræ fall together prematurely and confine that part of the tuberculous focus that is the cause of the paraplegia to the spinal canal—in other words, reproducing the state of affairs found in the spinal tumour syndrome? It is probable that shrinkage of the contents of the intraspinal lesion is likely to occur more readily in a case where it communicates with the large fusiform or globular abscess usually found lying anterior and lateral to the vertebral bodies. The natural safety-valve for an intraspinal abscess is the channel that runs between the diseased bodies; if this is closed early, absorption of the contents of the focus is likely to be very slow.

FIG. 545. *Case* S.7. Minimal bone destruction: paraplegia for seven years.

3. PARAPLEGIA IMPROVING, THEN RELAPSING AND REMAINING STATIONARY; OR MODERATELY SEVERE FROM THE FIRST AND SHOWING NO SIGNS OF IMPROVEMENT EVEN AFTER SEVERAL (4–7) MONTHS

Cases in this group belong essentially to Type I at first, and only too often gradually drift into Type II while the clinician is still fondly hoping that signs of recovery will ultimately appear. Every Type I paraplegia in which complete motor paralysis has been present for six months or rather less must be considered a possible Type II and should be investigated accordingly.

Good X-rays of the spine, including stereoscopic lateral views, should be taken and repeated if necessary at intervals of a few weeks. There are three possible explanations for the prolongation of severe paraplegia, and they will generally be revealed by the X-ray.

Pathological Dislocation (*see* Butler's article, p. 753).—The presence of this

condition was proven in four of my cases and one of Butler's. A typical case has also been recorded by the Sorrels[31] (p. 412), although the pathology of the condition was not clearly recognized by them.

Case S.37.—Complete motor paralysis in this patient with early active disease persisted unchanged for four months after she had been admitted to the Country Branch of the Royal National Orthopædic Hospital. She was therefore well on the way to becoming a Type II paraplegia. Serial X-rays showed a fusiform thoracic abscess steadily increasing in size, and I suspected that this was the cause of the persistent paraplegia. On April 6, 1933 (four months after admission) I performed a costo-transversectomy; the abscess cavity contained only a small quantity of pus and caseous material—hardly enough to convince one that the abscess was really the cause of the trouble. One month later the paraplegia was quite unchanged. Re-examination of the lateral X-rays showed that there was probably a pathological dislocation, and stereoscopic lateral views taken on May 2 showed the dislocation clearly (*Fig.* 546). This interpretation of the X-ray appearances was confirmed by Mr. Bankart. The spinal canal was explored on May 4, 1933, and the cord was found tightly stretched over a sharp bony ridge.

FIG. 546.—*Case* S.37. Lateral X-ray of the spine of a patient with paraplegia resulting from pathological dislocation. The sharp bony ridge in the floor of the spinal canal is clearly visible at A. It was still more evident in the stereoscopic X-rays.

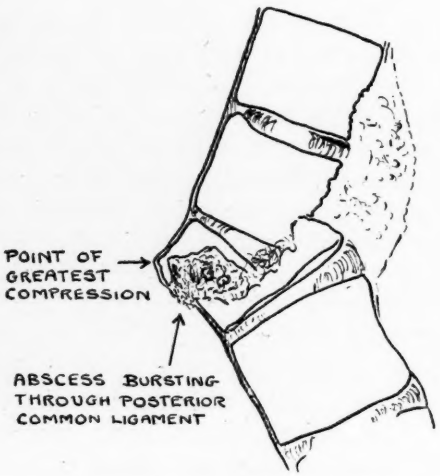


FIG. 547.—*Case* S.37. Diagram constructed from X-ray, operative, and post-mortem findings.

If the existence of this condition is borne in mind, it should not be difficult to recognize it in the investigation of Type II paraplegias.

Compression by a Sequestrum.—According to the Sorrels, the same severe persistent paraplegia may be caused by compression of the cord by a sequestrum. They have published an excellent example of this condition[31] (*Figs.* 548, 549), but they have not said how frequently a displaced sequestrum may be demonstrated radiographically. There are no cases of this type in my series, but a good one has been recorded by Butler (p. 757).

In pathological dislocation and in compression by a sequestrum, the prognosis is always hopeless as regards the paralysis, and generally so as regards life.

Continued Activity of the Disease.—This is by far the most frequent cause

of persistence of an early paraplegia. There are certain cases in which the patient's resistance is so low that the disease in the spine progresses relentlessly in spite of the most efficient conservative treatment, and if paraplegia is present, it will continue in a severe form for an unusually long time, unless spontaneous drainage occurs as a result of external rupture or internal tracking of the abscess. Not infrequently there is complete motor paralysis for more than six months, and by the time activity of the disease finally abates, the cord is permanently damaged, and recovery from the paralysis will be incomplete or entirely absent.

Fortunately the signs of continued activity are very definite and one or more of them is present in every case. They are irregular fever, wasting, radiographic evidence of extension of the bony lesion and increase in size of the abscess shadow, and sometimes the appearance of fresh tuberculous foci in other parts of the body.

In my series alone there were 8 cases of continued activity of the disease which ended with permanent paralysis, and 6 in which the recovery was delayed and incomplete. A number of these cases ended fatally—for the reason that the disease progressed unchecked and finally became generalized.

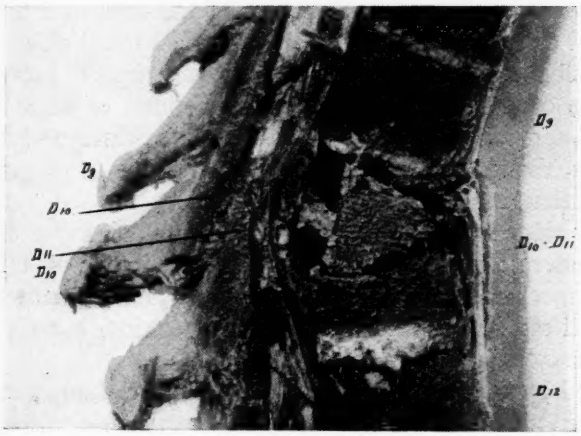


FIG. 548.—The specimen from a case of the Sorrels (Brun—Pauline, aged 52 years). The sequestra lie in the cavity of an active abscess, but it is the bony fragments and not the abscess that are responsible for the compression. It will be noticed that the posterior arch of D.10 has slipped down behind the arch of D.11; the spinal canal is therefore narrowed to some extent, a contributory factor in the compression of the cord.

FIG. 549.—An X-ray of the case shown in *Fig.* 548 taken during life. The large sequestrum is shown very clearly. (*Figs.* 548, 549 *are reproduced by kind permission of Professor E. Sorrel and Masson et Cie., Paris.*)

In posterior spinal disease with paraplegia, the condition may follow a Type I or Type II course, depending on the virulence of the infection. In one of my cases, S.36 (*Fig.* 540), the paraplegia was never complete and lasted for only eight months in all. In the other, S.64, the resistance of the patient was poor from the beginning, and paraplegia developed early in the course of the disease: voluntary control was soon lost, and never returned until a few months before the patient died of phthisis and amyloid disease, twenty-one months after he first came under observation.

TYPE III PARAPLEGIA

Type III paraplegia is immediately differentiated from Types I and II by the history and clinical picture.

The story is generally as follows—early in life the patient has suffered with spinal caries; as a result either of extensive disease or inadequate treatment, or both, bone destruction has been excessive, and a number of vertebral bodies, generally from four to nine, have been completely destroyed. The disease finally healed and the patient led a normal life; nothing unusual was noticed for a number of years apart from slowly increasing deformity of the spine, often so marked as to attract the attention of the patient and his relatives. Then after a long period, anything from four to forty years, the patient complains of a little weakness of the lower limbs or inco-ordination when walking: this marks the onset of the paraplegia. Pain is rarely felt, but if it is present, it takes the form of backache or irregular pains radiating down the lower limbs; only one patient in my series suffered with girdle pains.

On examination a severe deformity is found; local tenderness is generally absent—in fact there is nothing apart from the paraplegia to suggest active disease. The paraplegia is usually incomplete, but may be severe.

Butler has shown that in this large group of cases the majority are suffering from a recrudescence of disease in the neighbourhood of the spinal cord; in a smaller number there is evidence that points to stretching of the cord over a bony ridge in the floor of the spinal canal—a paraplegia of purely mechanical origin. It is most difficult, often impossible, to distinguish these two varieties of late onset paraplegia, but a consideration of the following points is sometimes of value:—

1. There may be clinical and radiographic evidence of active disease—further erosion of the vertebral bodies, the shadow of a recent abscess, or, as in one of my cases, S.23, a fresh abscess discharging on the surface.

2. The absence of a bony ridge in the floor of the spinal canal clearly excludes bony compression, but the converse is not always true: the presence of a ridge does not necessarily indicate mechanical compression. In one of my cases, S.22, a bony ridge was demonstrated radiographically and seen at operation, but the real cause of the paraplegia was renewed activity of the disease.

3. If the patient is treated by a method that tends to decrease the angulation of the spine (e.g., suspension on a Fisher frame), it would be reasonable to suppose that a paraplegia due to mechanical compression would either improve or, at least, remain stationary. If, in spite of such treatment, the paraplegia become steadily worse, the cause must be infective. In a recent case, S.110, a late onset paraplegia steadily increased in spite of suspension: at operation the spinal theca was found embedded in recent tuberculous granulation tissue.

The prognosis as regards life in late onset paraplegia is good. Even patients who become permanently paralysed rarely die as a result of the paralysis. The deformity of the spine and chest is probably a greater menace to life. Carey Coombs[6] pointed out the association of sudden fatal cardiac failure with severe kyphosis; but this is quite apart from the incidence of paraplegia.

The recovery rate in Butler's and my series of Type III paraplegias is as follows:—

	Under 16 Years			Over 16 Years		
	R.W.B.	H.J.S.	Total	R.W.B.	H.J.S.	Total
Type IIIa .. .. (recovered from paralysis)	24	11	35	10	12	22
Type IIIb .. .. (paralysis permanent)	6	6	12	13	7	20
Recovery rate	74·5 per cent			52·4 per cent		

Any patient under 16 years of age with late onset paraplegia has, therefore, an excellent chance of recovery—about 75 per cent. In later years the prospect is not so good—about 50 per cent.

We are unable to differentiate the prognosis in late onset paraplegia due to renewed activity of the disease from that due to compression by bone, seeing that in most of our cases it was impossible to be certain which of the two causes was operative.

Nor are we able to state in advance what chance of recovery there is likely to be in any particular case. The only reliable guidance is that given by the neurological features of the paraplegia (p. 770). Any case in which complete motor paralysis has been present for more than six months and the vibration sense lost is unlikely to recover completely. Butler has drawn attention to the composition of the cerebrospinal fluid in late onset paraplegia, and the possible association between a high protein content and a favourable prognosis. This observation may prove to be of value, but we have not yet examined a sufficient number of cases to justify any dogmatic statement.

SUMMARY OF PROGNOSIS

A guide to prognosis is given based on the neurological features of Pott's paraplegia, irrespective of the cause. It may be epitomized in the following way:

Very Favourable	Unfavourable	Hopeless
Paraplegia in extension	Sphincter involvement	Paraplegia in flexion
Voluntary control never lost	Urinary infection	Flaccid paralysis (except in cauda equina and brachial lesions)
Voluntary control absent for not longer than 6 months	Pregnancy	Loss of voluntary control for longer than six months
Absence of sensory loss		Loss of vibration sense
Absence of sphincter involvement		Old age

All these signs are of value in determining when a Type I paraplegia is changing to Type II, though they do not indicate the *reason* for the change. Similarly, they show when a Type IIIa case is likely to develop into a Type IIIb.

A clinical approach to the differentiation of Type I from Type II paraplegia is described. The histories of the various Type II paraplegias are highly characteristic.

A complete paraplegia of rapid onset is typical of concertina collapse of a vertebral body, or thrombosis of vessels supplying the cord: the two conditions are distinguished radiographically.

The spinal tumour syndrome is clinically unlike any other form of Pott's paraplegia; the only condition with which it may be confused is true spinal tumour.

A Type I case continuing into Type II—that is, remaining paralysed completely for six months or longer—is due either to pathological dislocation of the spine, or to compression of the cord by a sequestrum, or to continued activity of the disease: again, these three conditions are differentiated radiographically.

It may be objected that as several of the Type II conditions are so uncommon, too much attention has been paid to them in this paper, and that the suggestions that have been made are unlikely to be of real value in clinical practice. We must, however, bear in mind that these conditions, rare singly, when taken together

account for 21 per cent of all paraplegias occurring during the active stage of the disease, and they are responsible for many tragedies. Admittedly the outlook for the patient with Pott's paraplegia is good, but it might be better, and, as will be shown in the next section of this paper, a rational plan for the prevention of permanent paralysis can be based only on a careful analysis of the atypical cases, among which the Type II paraplegias form an important group.

It is possible that further investigation will establish other causes of Type II paraplegia. Several conditions described in the literature have been excluded by us on the grounds of insufficient evidence. We have confined our attention entirely to conditions in which the underlying pathology has been definitely established.

As regards Type III paraplegia, we are still very much in the dark both in our methods for differentiating the "renewed activity of disease" type from the less common type with bony compression of the cord, and also in giving a reliable prognosis.

A few signs are of value, but they are present in only a minority of the cases.

Part II. PREVENTION AND TREATMENT

TYPE I PREVENTION

Seeing that paraplegia is not infrequently the first clinical sign of spinal caries, the prevention of Type I paraplegia is not always possible. This is particularly the case in adults, in whom the disease often continues for many months with relatively little bone destruction, no deformity, and few symptoms. However, early symptoms in the form of local or referred pain are generally present, and too often overlooked by the clinician. Spinal caries as a cause of backache in adults is insufficiently recognized. It seems as if *deformity* stands in the foreground of the commonly accepted clinical picture of the disease. Actually, in adults, obvious deformity is rare and pain almost invariable. In two of my cases, both adults, upper abdominal pain led to diagnoses of gall-stones in one case, and duodenal ulcer in the other. The first was treated by cholecystectomy, the second by dieting. It was not until paraplegia developed that the real cause of the pain was discovered.

In children, on the other hand, paraplegia as a first sign is rare. The deformity due to bone destruction develops early and rarely escapes recognition, and backache —uncommon in children—immediately suggests a serious spinal lesion.

Nor in the established case of spinal caries is the prevention of Type I paraplegia always possible. By pure ill-luck, the primary focus may lie far back in a vertebral body, or in a lamina, and spread rapidly into the spinal canal. In other cases the virulence of the infection may be such that the patient never has the slightest chance of recovery: the disease spreads rapidly, the cord is inevitably involved, and the patient soon succumbs to the generalized disease.

As far as we know, there is only one special measure applicable during the course of ordinary conservative treatment that is of definite value in diminishing the incidence of paraplegia—namely, hyperextension of the spine.

It is certain that extensive destruction, and probable that the formation of large abscesses, occur more readily in any case of joint tuberculosis in which movement, particularly friction between joint surfaces, is allowed during the stage of activity. In hip disease we know that bone destruction and abscess formation are reduced

to a minimum by combined traction and fixation. The same is true of spinal caries. In the spine firm fixation is not difficult to maintain, but it is impossible

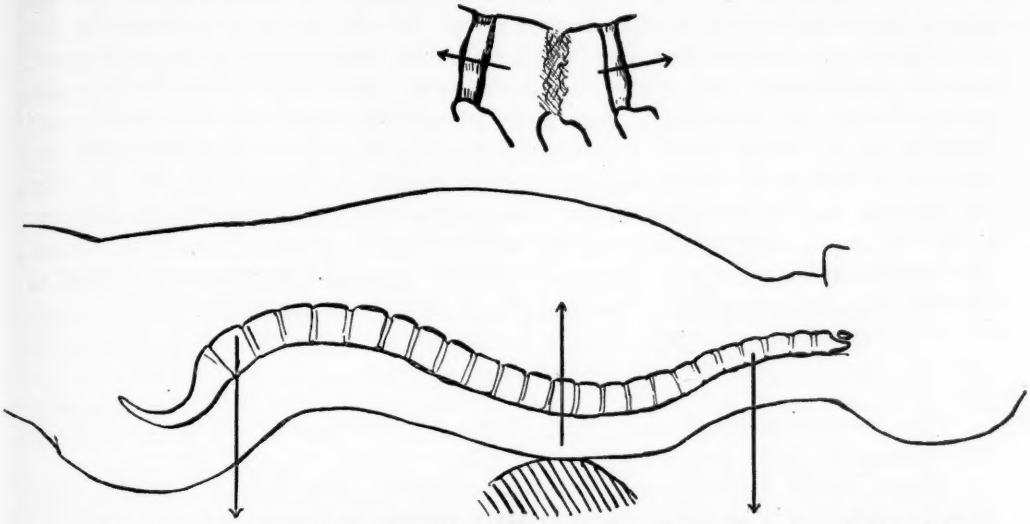


FIG. 550.—The simplest form of hyperextension of the spine. Pressure is made at the site of the lesion, with the patient lying supine. The extending force is the body weight acting above and below the lesion. There must always be a pressure point at the level of the lesion. If this is absent, as in the hyperextended anterior shell, hyperextension results only in the development of compensatory curves.

to apply efficient longitudinal traction to the thoracic spine where bone destruction is often so marked. However, the same effect may be obtained without difficulty by hyperextension, provided that constant pressure is applied at the level of the lesion (*Fig.* 550). When the thoracic spine in a case of active disease is hyperextended, the involved vertebræ tend to separate; not infrequently radiographically visible separation actually occurs. In any case the vertebræ are relieved from longitudinal pressure, and the grinding resulting from the constant respiratory movements is reduced. I have been able to observe the amount of bone destroyed in a large number of cases of thoracic caries in children, some of whom were treated in plaster beds, and others in hyperextension. In the latter the bone loss was considerably less than in the first group. Hyperextension is only necessary for as long as there is radiographic evidence of active

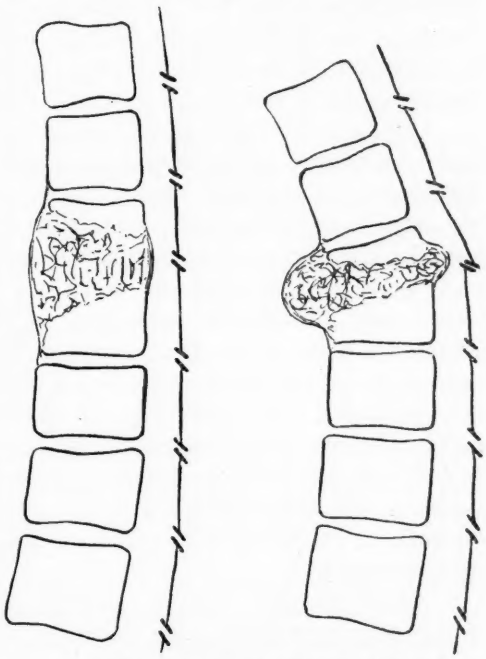


FIG. 551.—It is possible that when inflexion of the spine occurs in an active case of caries, the contents of the abscess are squeezed circumferentially, and in encroaching on the spinal canal may compress the cord. Probably a reversal of this process accounted for the rapid recovery in a number of Goldthwait's cases of paraplegia that were treated by gentle hyperextension of the spine under anæsthesia.

spreading disease. As soon as activity has abated, hyperextension should be discontinued and the diseased vertebræ allowed to fall together. Healing will then occur with the loss of fewer vertebral bodies than would have been the case under treatment in simple recumbency. Furthermore, it is probable that the abscess so constantly present is favourably influenced by hyperextension both as regards size and shape. Goldthwait[14] found that if a patient with active disease and paraplegia was anæsthetized, extension of the spine could easily be obtained by gentle pressure on the kyphos, and not infrequently the paralysis disappeared, often with remarkable rapidity. He was unable to offer any explanation for this phenomenon. It can only be explained by the influence of the extension of the spine on the shape of the abscess (*Fig.* 551), and if this suggestion is correct, it furnishes another powerful argument in favour of hyperextension of the spine in the prevention of Type I paraplegia.

TYPE I TREATMENT

The treatment of almost every form of Pott's paraplegia is primarily conservative.

Fixation.—If treatment in hyperextension is called for in uncomplicated thoracic disease, it is all the more necessary if paraplegia is present. Any apparatus complying with the conditions already described (*see Fig.* 550) may be used. In my experience the Bradford frame is most satisfactory. Adults who cannot tolerate frame treatment may find a hinged plaster bed more comfortable. Heavily padded frames of the Robert Jones pattern, while providing excellent fixation, are not to be recommended in cases of thoracic disease, on account of the difficulty of ensuring that the spine is adequately hyperextended.

Unfortunately, the paraplegia is sometimes so severe that treatment in hyperextension cannot be continued on account of the rigidity of the lower limbs and the tendency to trophic sores. Such cases must be nursed in an ordinary plaster bed, or, if very severe, in a reversible spinal bed. In mild cases the lower limbs may be left free. The moment spasticity becomes marked, adhesive extension must be applied to the lower limbs and long metal splints to hold the feet at right angles and the knees in extension. It is advisable to have the splints bent to an angle of 10° or 15° at the knees; full extension is uncomfortable and may lead ultimately to the development of back-knee. Fixation of the lower limbs must be maintained until spasticity has almost disappeared. In the most severe cases extensions and splints are not tolerated. The spasm is uncontrollable, and if efforts to keep the lower limbs straight are continued, severe trophic sores are certain to develop. In these cases the lower limbs must be left free, and every joint put through its full range of movement twice a day in order to prevent the development of contractures.

Acute retention requires prompt catheterization under rigidly aseptic conditions. Continued retention is uncommon, and I have never found it necessary to catheterize a patient repeatedly, or evacuate the bladder by manual expression as recommended by the Sorrels. In all cases with bladder involvement, it is wise to administer some form of urinary antiseptic from the first, whether infection is present or not. Severe constipation should be controlled by enemata.

Massage has no place in the treatment of established paralysis unless there

is a lower motor neuron lesion. In the ordinary type of spastic paraplegia, physical treatment is called for only when the paralysis is well on the way to recovery; at this stage massage and exercises are of great value in getting rid of the last traces of inco-ordination and clonus. In lower motor neuron lesions, where muscular wasting is present from the first, the treatment is the same as in a case of infantile paralysis during the stage of recovery.

Operation.—Although spontaneous recovery is assured in almost every Type I paraplegia, there are certain cases bordering on the Type II "continued activity of disease" group, in which the paralysis is so profound and prolonged that the patient is reduced to a condition of extreme misery and the surgeon to a state of constant anxiety. In these cases operation is of value.

It has frequently been observed that if an abscess appears superficially in a case of active disease with paraplegia, the latter begins to clear up rapidly as soon as the contents of the abscess have been evacuated. There is an undoubted connection between drainage of the focus of disease and relief of the paralysis: several useful procedures which may be classed together as lateral drainage operations have been evolved from this observation.

LATERAL DRAINAGE OF A SPINAL ABSCESS: COSTO-TRANSVERSECTOMY.—Although others before Ménard[20] had performed this operation, he was the first to demonstrate its value in Pott's paraplegia. By 1900, when he published his famous book, *Etude pratique sur le Mal de Pott*, he had performed costo-transversectomy 23 times. In 13 cases recovery commenced immediately after the operation, in 4 after one week, and in 2 at the end of three months. There were 5 failures, 2 due to compression by bone, and 3 in which the cause of failure was unknown.

Operative Technique.—According to Ménard, the operation should be performed on the left side. A transverse incision, 5 to 7 cm. in length, is made over the vertebral end of the rib corresponding to the apex of the kyphos. After the rib has been exposed, the periosteum is carefully elevated on the superficial and deep surfaces. The rib is divided with nibbling forceps at a point about 4 cm. from the tip of the transverse process of the corresponding vertebra. The transverse process is then cut through at its base and removed after its costal attachment has been divided. The inner end of the rib is now lying in a periosteal tube attached only by its head. It is removed by combined twisting and traction. Sometimes the removal of the rib opens the abscess cavity. If it does not, the tunnel should be gently explored with the finger, and when the wall of the abscess is located it is incised with a blunt instrument.

Ménard does not mention ligation and division of the neurovascular bundle in a case where more than one rib has been resected. I have found that a most satisfactory exposure is obtained if the neurovascular bundle is encircled with a double ligature, tied in two places, and divided.

When it is proposed to resect more than one rib a longitudinal incision in the line of the costo-transverse joints may be employed with advantage.

ASPIRATION OF A THORACIC ABSCESS.—This form of treatment was introduced as an alternative to costo-transversectomy, the great virtue claimed for it being that the risk of secondary infection is almost entirely eliminated.

In 1908 Finck[10] reported the successful aspiration of a mediastinal abscess.

Schede[27] describes a most exact method for determining the depth of an abscess. The necessary calculation is distinctly complicated, but he claims that

if the trocar and cannula are introduced in the way that he describes, nothing but the abscess will be encountered.

Calvé[3] believes that inflexion of the vertebral column at the site of a thoracic lesion squeezes an abscess laterally and posteriorly. It is, therefore, a mistake to hope to relieve tension in the part of the abscess that is compressing the cord by introducing a trocar and cannula along the side and to the front of the vertebral bodies. He introduces the instrument through the intervertebral foramen into the abscess as it lies directly anterior to the cord. Though the method may be sound in theory, most of us would say with Delbet, " Je n'ai pas cherché à en évacuer par la technique de Calvé qui me partait au-dessus de mes forces."

The most attractive method is that described by Valtancoli.[37] He takes antero-posterior and lateral X-rays of the spine in the usual way, and then with barium in the œsophagus. Having localized the abscess exactly, he introduces a slightly curved trocar and cannula. The instrument must not be less than 9 cm. in length, and the bore of the cannula not less than 3 mm. The instrument is passed close to the upper border of the rib in order to avoid the intercostal vessels, then towards the mid-line, aiming directly at the vertebral body. Pus is usually found at a depth of from 6 to 8 cm.

The disadvantages of mediastinal aspiration are obvious. Puncture of the lung and even the aorta (!) have been reported, but no one confesses to any serious trouble having followed such accidents.

It is a matter of history that Ménard finally abandoned costo-transversectomy on account of the fatalities that resulted from infection of the sinus that he deliberately produced by operation. However, those who have followed him have not all had the same unfortunate experience: Jackson Clarke[5] (1903), Wassiliew[40] (1909), Steindler[34] (1929), and Girdlestone[13] (1931) have expressed themselves in favour of the operation and have not considered secondary infection a serious menace. Indeed, it never occurred in most of the cases that these observers have recorded. No doubt this was due in part to the practice (of Girdlestone, for example) of suturing the wound after evacuation of the abscess, but the real secret of their success was that adequate conservative treatment was continued for many months after the drainage operation had been performed. As Girdlestone so properly insists, costo-transversectomy is only an incident in the treatment. Most of Ménard's patients were allowed to walk the moment the paraplegia had cleared up, and in the light of our present knowledge it is quite evident that they were never given a real chance to recover from the active disease in the spine.

We are of the opinion that costo-transversectomy is of very definite value in certain cases of paraplegia of early onset: it is probably the best of all the operations that have been proposed and practised.*

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* *Laminectomy.*—Before 1900, Ménard had reached the conclusion that laminectomy was of very limited value in the treatment of Pott's paraplegia. His views have been upheld so repeatedly in papers written since that time, that a detailed discussion of the operation is no longer necessary. Unfortunately, in spite of the mass of adverse evidence, laminectomy for severe Pott's paraplegia still finds a place in the teaching and practice of many surgeons. As will be shown later, the operation may be of some value in certain rare forms of paraplegia, but as a routine procedure it is to be condemned. *See Table II*, p. 796.

Spinal Fusion.—De Courcy Wheeler[41] and others are of the opinion that patients with paraplegia recover more rapidly after a fusion operation has been performed. The evidence

The advantages claimed for costo-transversectomy are :—

1. It attacks the main cause of the paraplegia—the abscess arising from the focus of disease in the spine.

2. Emptying of the abscess cavity reduces the pressure on the cord and may also reduce the toxicity of the focus—which, as Butler has shown, is probably of considerable importance in determining changes of conductivity in the cord.

3. After costo-transversectomy, drainage of tuberculous material is away from the cord—not around it, as after laminectomy.

4. The operation does not weaken the bony spine, as does laminectomy.

5. The technique is not particularly difficult, nor is there any great operative risk.

6. A relatively large drainage canal is created, through which it is possible to evacuate not only fluid pus, but granulation tissue, bone sand, and caseous material. This cannot be accomplished by any of the methods of aspiration that have been described.

7. Any further collection of pus that may form after costo-transversectomy will come to the surface at the site of operation. If the wound has been sutured, aspiration may safely and easily be performed at a point just to one side of the incision.

If a sinus forms, it should be regarded as beneficial rather than dangerous. It indicates that continuous drainage is needed for a longer or shorter period, and the sinus will close as soon as the lesion in the spine begins to heal. Secondary infection did not occur in any of our cases.

Drainage of a cervical abscess causing pressure signs is exactly analogous to costo-transversectomy, and need not be described separately.

It has already been shown that owing to continued activity of the disease, a certain number of cases that commence as Type I end as Type II; compression and intoxication of the cord continue for so long that irreparable damage is inflicted. It is therefore necessary to consider together the operative treatment of Type I and the treatment of Type II due to continued activity of the disease.

Costo-transversectomy is indicated in profound Type I paraplegia. Though recovery may possibly be expected to occur spontaneously, there is no reason why the patient should be allowed to suffer for many months if a simple and safe operation can give him relief.

Case S.24.—A girl, 8 years of age, was admitted to the Country Branch of the Royal National Orthopædic Hospital in July, 1929, with spinal caries of about four months' duration. The X-ray showed an early lesion of Th.9–10 with a small fusiform abscess.

November, 1929.—X-ray, abscess larger.

October, 1930.—Onset of paraplegia.

During the following eleven months the paraplegia became more and more profound until the limbs were held rigidly in extension, voluntary power was lost, and sensation (except vibration) absent below the level of Th.10. Serial X-rays showed a steady increase in size of the abscess. It was decided that costo-transversectomy should be performed, but the parents obstinately refused to give their consent.

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supporting this view is not convincing, and in America, where fusion operations are often performed early in the course of the disease, many cases have been found (by Steindler and Silver, for example) to develop severe paraplegia after the performance of a fusion operation. Two cases in my series, Nos. S.45 and S.75, developed paraplegia after 'successful' fusion operations had been done. The value of the fusion operation in cases with paraplegia has not yet been proved.

February, 1932.—Patient removed from hospital against advice, and nursed at home. During the following two years she was visited regularly.

August.—A large abscess appeared below the left 12th rib.

October.—Abscess ruptured, discharging at least a pint of pus. Almost immediately the paraplegia began to clear up.

September, 1934.—Good voluntary power, but still a little spasticity.

In this case, fortunately for the patient, spontaneous drainage occurred, but it is clear that had operation been performed in 1931, she would have been spared twelve months' misery. Furthermore, it is probable that the paralysis would have cleared up completely after operation, whereas now the lower limbs are still a little spastic. The following case provides a happy contrast.

Case S.38.—A girl, 12 years of age, was admitted to the Royal National Orthopædic Hospital (Great Portland Street) in September, 1922, under the care of Mr. Jackson Clarke, with two months' history of trouble in the back and weakness of the lower limbs. On admission she was found to have an early lesion of Th.6–8, and well-marked but not severe paraplegia.

November, 1922.—Complete loss of voluntary power, anæsthesia below Th.8, and incontinence of urine.

Dec. 4.—Costo-transversectomy (Mr. Jackson Clarke). Large abscess opened.

Dec. 19.—Complete sensory recovery, a little voluntary control of the toes. No incontinence.

Dec. 27.—Wound soundly healed. Transferred to Country Branch, Stanmore; conservative treatment continued.

During the following two years, the abscess reappeared and was aspirated repeatedly. The paraplegia cleared up completely.

September, 1925.—Discharged, walking well. Condition since this date most satisfactory.

This is an example of a perfectly managed case. As soon as it became evident that the paraplegia was certain to be severe and prolonged, operation was performed. As a result the patient was completely paralysed for only two months. The costo-transversectomy ensured that further collections of pus would make their way to the surface and not towards the spinal canal: they were dealt with successfully by aspiration. Conservative treatment was continued for an adequate length of time after operation.

Clearly, then, we are not justified in temporizing with any severe Type I paraplegia, for it may develop into a hopeless Type II case. Operation should not be delayed for longer than six months after voluntary control has been completely lost,* provided that the X-ray shows a definite abscess shadow. It has already been shown that spontaneous recovery after more than six months of complete motor paralysis will be either incomplete or entirely absent.

Several of my cases were operated on too late—eighteen months, two years, or even three years after the onset of complete motor paralysis—in no case with the slightest benefit (*see Table I*, p. 795).

A further indication for operation is evidence of pressure on other vital structures—with or without paraplegia. In one of our cases, a large cervico-mediastinal abscess was the cause of severe paraplegia and marked respiratory obstruction. Complete relief rapidly followed incision and evacuation of the abscess.

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* It must be emphasized that paraplegia of only six months' duration is not necessarily an indication for operation. Paraplegia may be present for as long as two years, and yet voluntary control may never have been completely absent.

TYPE II PREVENTION

From all that has been said of the pathology of concertina collapse of a single vertebral body, pathological dislocation, and compression of the cord by a sequestrum, it is clear that these conditions can generally be prevented by prolonged hyperextension of the spine. This is in fact the only non-operative measure of any value in the prevention of Type II paraplegia, but unfortunately the condition of the patient is sometimes so critical that this ideal method of nursing cannot be employed—and only too often the accident that damages the cord beyond hope of recovery has occurred before the patient comes under treatment.

TYPE II TREATMENT

In thrombosis of vessels supplying the cord and in concertina collapse of a vertebral body, no radical treatment is of any avail. Early laminectomy is indicated in all cases of spinal tumour syndrome, though it has yet to be shown that the condition may be completely cured by surgical intervention. Costo-transversectomy is indicated in all cases with continued activity of the disease, and there is abundant evidence to show that recovery will occur, provided that the

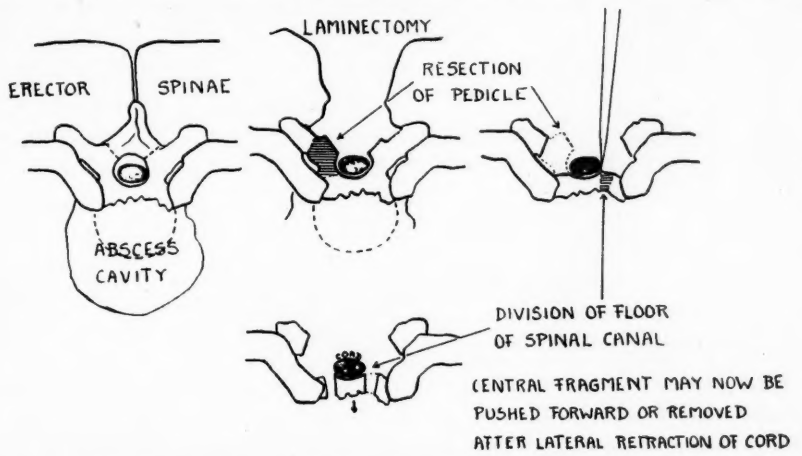

LAMINECTOMY
RESECTION OF PEDICLE
ERECTOR SPINAE
ABSCESS CAVITY
DIVISION OF FLOOR OF SPINAL CANAL
CENTRAL FRAGMENT MAY NOW BE PUSHED FORWARD OR REMOVED AFTER LATERAL RETRACTION OF CORD

FIG. 552.—In pathological dislocation the cord is stretched over a sharp ridge in the floor of the spinal canal. The ridge is formed by the remains of a vertebral body which is part of a more or less complete bony ring encircling the spinal cord. In order to decompress the cord the ring must be broken anteriorly. The last step in the operation is not shown—division of the one intervertebral disc that holds the offending fragment to its neighbouring vertebral body. This should present no technical difficulties.

operation is performed early and that the patient does not succumb to generalized tuberculosis. The details of treatment have already been discussed.

No case of compression by sequestrum has yet been dealt with successfully by operation. Theoretically, it would seem possible to remove even a large sequestrum from the front of the cord by a large costo-transversectomy approach. In *Case* S.58 a sequestrum was thought to be the cause of a severe persistent paraplegia: a dense irregular shadow was visible in stereoscopic lateral X-rays, lying immediately anterior to the cord in the centre of an extensive tuberculous focus. After resection of two ribs and ligation of the intervening neurovascular bundle, a good exposure of the focus was obtained. The 'sequestrum' proved to be a large dense mass of caseous material, which I removed without difficulty. If

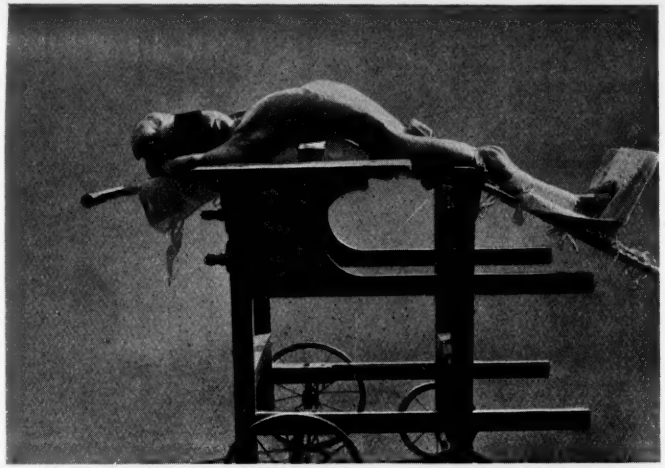
the mass had been a sequestrum, it could probably have been removed just as easily, and I see no justification for the pessimism of the Sorrels about this condition.[31] The primary difficulty is in recognizing the presence of a sequestrum at an early date.

In pathological dislocation radical operative treatment may hold out some prospect of relief, and although I have no records of successful operative intervention, it must be evident that an operation based on a clear understanding of the morbid anatomy of the condition is at least worth a trial.

As the fragment of bone responsible for compression of the cord is part of a vertebral body, it must at the same time form part of a bony ring encircling the spinal canal. The integrity of this ring will depend upon the extent to which the pedicles have been eroded by the disease. If the anterior half of the ring could be resected, the cord would be liberated, and this should be the aim of any operation designed for the relief of this condition. The steps of such an operation are shown in *Fig.* 552. In *Case* S.37 (*see Fig.* 546) I performed a laminectomy and found the cord stretched over a bony ridge in the floor of the spinal canal: the right pedicle attached to the ridge was resected and an attempt was made to remove the latter. This step appeared to be successful, and the theca, which up till that moment had been white, tense, and pulseless, became pink, lax, and pulsatile. Unfortunately, there was no improvement in the paraplegia after operation, and the patient died of acute tuberculous pneumonia and sepsis from sores seven weeks later. At the autopsy it was found that the bony ridge had been incompletely removed (*Fig.* 553); the left half was still present and the cord stretched over it. It is probable that the angulation increased after the operation, and that an incomplete removal of the ridge, apparently adequate at the time of operation, did not afford permanent decompression of the cord. It is on this failure that the suggested operation is based. Due consideration has been given to the possibility of a postero-lateral attack on the ridge by an approach resembling a costo-transversectomy, but as the ridge may be attached to both of its pedicles, it must be clear that the only reasonably safe and sure method of removal of the triangular fragment is in the way suggested.

FIG. 553.—*Case* S.37. The cord and the remains of the bony ridge, which is compressing the cord on its left side.

Among German surgeons—Tillmanns,[36] Borchers,[2] and Nureddin,[23] for example—there is remarkable unanimity in advising laminectomy for disease commencing in the neural arches—posterior spinal disease. Operation is indicated even in the absence of paraplegia. It is the surgeon's one opportunity for performing a complete excision of the diseased bone in a tuberculous spine. If a radical



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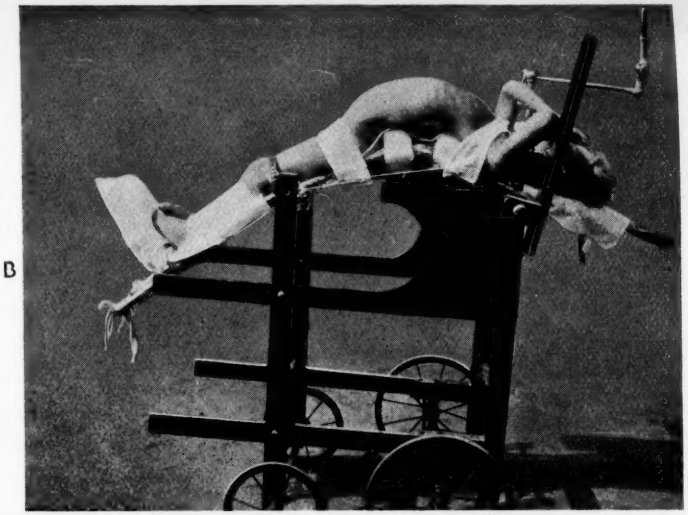

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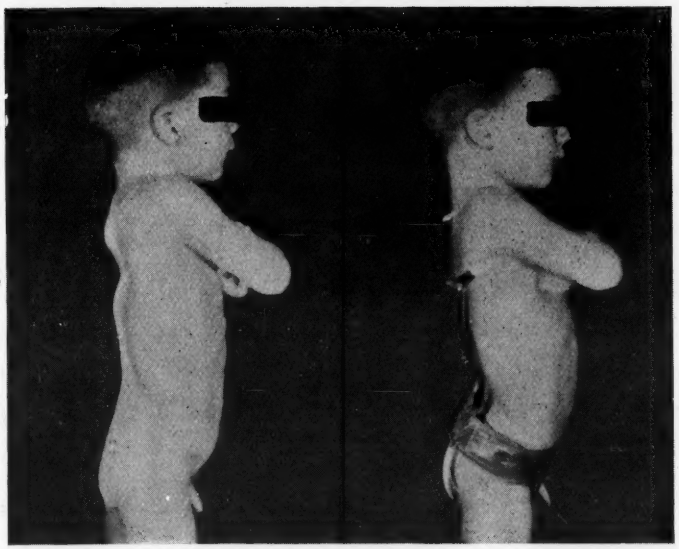

C

FIG. 554.—An example of the production of a well-balanced spine as the end result of treatment of extensive low thoracic disease in a child. A, T.R., aged 4, at onset of disease. No compensatory curves. B, Two years later. The kyphos now falls into the hollow between the two cross pads of sponge-rubber and there are excellent compensatory curves above and below. C, After three years in recumbency (two and a half years with the cross padding) the general balance of the spine is excellent. He is seen standing with and without a Jones' back brace. (*Patient under the care of Mr. R. W. Butler, to whom I am indebted for the photographs and X-ray—Fig.* 555.)

operation is not performed, infection of the bodies will almost invariably follow, as in two of my cases. Successful operations have been recorded by Sharpe[29] and Pels-Leusden,[25] though in the case reported by the latter a lesion appeared later in the bodies of Th.12 and L.1, in spite of a complete excision of the initial focus in the arch of L.1.

TYPE III PREVENTION

Late onset paraplegia occurs only after severe and extensive disease of the thoracic spine which has led to the development of considerable deformity. The

FIG. 555.—X-ray of spine of patient shown in Fig. 554.

prevention of this very common variety of paraplegia is dependent upon the efficiency of the treatment of the active disease. The frequency of late onset paraplegia (accounting for about 50 per cent of all cases) is a reproach on the management of spinal caries in this country.

The essentials of efficient treatment are :—

1. Reduction of bone destruction to a minimum. This is best accomplished by nursing the patient in some form of hyperextension apparatus.

2. Prolonged treatment in recumbency until long after bone destruction has

ceased and signs of repair are apparent radiographically. Again it must be emphasized that in most cases the cause of late onset paraplegia is renewed activity of the disease. In a number of our cases it was very evident that the disease had never really healed: the patients were not kept on their backs for a sufficient length of time. In some cases four years' continuous treatment is essential.

3. When bone destruction has come to an end, simple hyperextension—which holds the focus open—should be discontinued and the involved vertebræ allowed to fall together and fuse. At the same time compensatory curves must be induced above and below the lesion, so that the patient will have a balanced spine when he gets up (*Figs.* 554, 555). The nearer the centre of gravity approaches to the spinal column, the less leverage will there be at the site of the healing lesion, and the more the compressive force tending to bring the involved vertebræ firmly together, so encouraging fusion.

4. At this stage some form of fusion operation may be performed to strengthen the weak point, but before this is done the involved vertebræ must be in good contact with each other, and compensatory curves must have been produced.

Late onset paraplegia rarely occurs after efficient treatment of the active and healing tuberculous spine.

TYPE III TREATMENT

The treatment of late onset paraplegia is essentially conservative. Prolonged rest in recumbency at an open-air hospital is necessary, as there is usually a recrudescence of active disease in the spine. Hyperextension is of very doubtful value, and there are no rational grounds for employing it.

In a certain number of cases (25 per cent in children, 50 per cent in adults) it becomes evident, as treatment is continued, that recovery is unlikely to take place with purely conservative measures. The question of operative intervention must be considered. We must remember that these cases are bad risks—whatever operation, spinal or abdominal, is performed on them. There is gross deformity of the chest, the vital capacity is diminished, and there is generally some degree of cardiac inefficiency. As late onset paraplegia is rarely fatal of itself, operation should therefore be advised only with great caution.

If a fresh paravertebral abscess is clearly demonstrable, costo-transversectomy is indicated (*Table I, Case* B.45—p. 795), though generally the reactivated focus is confined to the spinal canal.

Laminectomy is usually the only hope, but there is a danger peculiar to this operation. Examination of many specimens from cases of late onset paraplegia shows that the integrity of the spinous processes and laminæ (which are often solidly fused) is sometimes the only factor preventing gross dislocation of a severely damaged spine. If laminectomy is performed (without subsequent grafting), the bony spine is virtually transected and subluxation may occur, and so lead to even more extensive damage to the cord than was present before operation. In some of our cases this actually happened, either soon after operation or when the patient was allowed to sit up in a support, and the last state of that man is worse than the first. Quite apart from this risk, the removal of epidural granulation tissue or simple decompression of the cord for late onset paraplegia is not often followed by recovery. Operation was successful in only 2 out of 9 of our cases. Radical treatment may sometimes be justifiable, but only when requested by the patient or his

relatives after the poor chances of success have been fully explained. The operation itself should always be combined with grafting.

It is possible that operation will prove more successful in cases where the cord is stretched over a bony ridge. Simple laminectomy is futile (*see Fig.* 538 and *Table II, Cases* S.43 and S.100—p. 797). Nothing less than the removal of the hump over which the cord is stretched will suffice.

In *Case* S.82 there was good reason to suppose that bony compression was the cause of a late onset paraplegia (*Fig.* 556). The patient was at first under my care,

FIG. 556.—*Case* S.82. A woman of 42 years. Apparently healed thoraco-lumbar caries; paraplegia of gradual onset occurring about thirty years after the disease had been active. Lateral X-ray, taken after injection of lipiodol via cisterna magna, shows incomplete thecal block—probably due to projection backwards of bony spur at A.

but was transferred later to Mount Gold Hospital, Plymouth, under the care of Mr. Norman Capener. I suggested to him that if the paraplegia did not clear up under conservative treatment, operation might be worth considering. Some months later he wrote to say that as the woman's condition was not improving, he had explored the spinal canal. His account of the operation is as follows:—

Curved incision to right of kyphos. Skin-flap turned medially. At apex of kyphos right sacrospinalis divided transversely and retracted upwards and downwards. Laminæ, transverse processes of three or four vertebræ stripped subperiosteally on right side only. The two central transverse processes were then excised together with the lateral portions of the laminæ almost to the roots of the spinous processes: corresponding pedicles resected. The intervening nerve-trunk, which appeared to be the first lumbar, was divided about ¾ in. from the theca and used as a means of rotating the theca backwards. On account of the deformity, the bodies of the vertebræ came well into view and the region of the posterior and right lateral surfaces was readily explored. Absolutely no evidence of active tuberculous disease was found. At the apex of the deformity the intervertebral spaces could be recognized but were apparently completely ossified. There seemed no doubt that the lesion was

Table I.—RESULTS OF COSTO-TRANSVERSECTOMY

Case No.	Type	Age	Duration of Paraplegia before Operation	Operative Findings	Result		Comments
					Life	Paraplegia	
B.42	I	17	7 months	Large abscess: much pus and caseous material	Recovered	Recovered	Sinus formed: closed later
B.44	I	47	5 months	Much pus and caseous material	Recovered	Recovered	
B.46	I	38	4 months	Much pus and caseous material	Recovered	Recovered	
B.48	I	26	3 months	No abscess: very active tuberculous granulation tissue	Recovered	Permanent	Operation should not have been performed: no radiographic evidence of abscess
S.38	II	12	3 months	Large abscess	Recovered	Recovered	Many aspirations afterwards, continued activity of the disease
S.58	II	14	6 months	Much caseous material and bone sand	Recovered	Recovered	Sinus formed and discharged for many months after operation. Continued activity of the disease
S.46	II	3	6 months	Large cervico-mediastinal abscess bulging into pharynx	Recovered	Recovered	Severe dyspnœa in addition to paraplegia. Cervical abscess opened. Sinus formed and closed later. Continued activity of the disease
S.49	I	16	2 months	Large cervical abscess	Recovered	Recovered	Evacuation of abscess through cervical incision
B.45	IIIa	37	4 months	Much pus and caseous material	Recovered	Recovered	Sinus formed: closed later
B.47	II	28	4 months	Large abscess	Recovered	Recovery then relapse	Laminectomy later—failure: a second costo-transversectomy should have been performed. A definite case of continued activity of the disease
S.31	II	11	13 months	Thick walled abscess containing solid caseous material	Died	Permanent	Operation delayed too long. Laminectomy was performed after the costo-transversectomy. Death eight days after operation. A case of continued activity of the disease
S.41	II	10	2 years 2 months	Thick caseous material and bone sand	Survived	Permanent	Operation delayed too long. Continued activity of the disease
S.102	II	12	2 years	Much caseous material	Survived	Permanent	Operation delayed too long. Continued activity of the disease
S.37	II	15	7 months	Very little caseous material and pus	Died tuberculous pneumonia	Permanent	Pathological dislocation cause of paraplegia. Costo-transversectomy should never have been performed

Table II.—RESULTS OF LAMINECTOMY

CASE NO.	TYPE	AGE	DURATION OF PARAPLEGIA BEFORE OPERATION	OPERATIVE FINDINGS	RESULT		COMMENTS
					Life	Paraplegia	
B.49	I	46	About 8 months	Prominent mass of tuberculous débris and bone anterior to cord; dura very vascular and covered with tuberculous granulation tissue	Recovered	Recovered	Inadequate treatment before operation; laminectomy performed just as good conservative treatment was commenced. Recovery would probably have occurred apart from operation
S.31	I	11	1 year 2 months	Cord compressed against laminæ by abscess	Post-operative death	?	
B.50	II	40	4 months	No pulsation of cord opposite Th. 8	Died	Permanent	
S 14	II	23	3 years 4 months				Minimal bone destruction (*see* p. 777), operation should have been performed earlier
S.34	II	30	1 year 5 months	Apparently healthy pulsating theca pushed back to some extent; further exploration of spinal canal abandoned on account of patient's poor condition	Died	Permanent	Continued activity of disease
S.44	II	9	6 months	Cord pushed back, and partially surrounded by granulation tissue	Survived	Temporary recovery then permanent	Continued activity of disease
S.66	II	29	1 month		Died	Permanent	Very active disease
B.56	II	25	3 years	Cord tightly stretched over a large angulation	Survived	Permanent	Pathological dislocation
S.37	II	15	7 months	Cord tightly stretched over angulation: removal of bony ridge attempted	Died	Permanent	Pathological dislocation
S.62	II	16	3 months	Abscess surrounding cord which was stretched over a bony ridge in floor of spinal canal	Died	Permanent	Pathological dislocation

S.93	II	16	1 month	Granulation tissue in spinal canal : cord stretched over angulation	Died	Permanent	Pathological dislocation
S.67	II	37	2 months	Granulation tissue and abscess	Died	Recovered	Spinal tumour syndrome
S.80	II	21	1 month	Granulation tissue	Survived	Permanent	Spinal tumour syndrome
S.61	IIIa	37	2 months	Abscess anterior to cord	Recovered	Recovered	
S.22	IIIa	10	6 months	Granulation tissue around cord	Recovered	Recovered	Postero-lateral approach : laminæ preserved
S.82	IIIa	42	8 months	Bony ridge in floor of spinal canal removed	Recovered	Recovered	Postero-lateral approach : laminæ preserved
B.51	IIIb	27	6 months	Granulation tissue and caseous material removed from epidural space : pulsation returned to cord	Survived	Slight improvement	
B.54	IIIb	35	1 year	Epidural space full of dense granulation tissue	Survived	Slight improvement	
B.55	IIIb	44	4 to 5 years	No evidence of pressure by bone and no active tuberculous material found	Survived	Slight improvement	
B.57	IIIb	27	3 months	Cord buried in caseous material and pus, most of which was removed : pulsation in cord did not return	Survived	Permanent, worse after operation	
B.58	IIIb		2 years	Abscess pushing cord backwards : contents removed	Survived	Improved, then permanent	Subluxation of spine when patient got up
S.32	IIIb	26	2 years	Granulation tissue	Survived	Permanent	
S.43	IIIb	12	5 months	Cord stretched over bony ridge	Operative death	?	
S.100	IIIb	14	1 year 10 months	Cord stretched over large bony ridge	Died	Permanent	
S.110	IIIb	9	8 months	Granulation tissue	Survived	Worse	

quite healed. After the cord had been rotated and the extradural stripping of the posterior surface of the bodies carried inwards anterior to the cord, a definite bony spur was encountered. During the stripping of this spur sharp spasms of the legs were produced. The spur appeared to be projecting from the posterior inferior edge of a vertebral body slightly cephalic to the actual apex of the kyphos. This spur was cut away with a chisel, the muscles were drawn together, and the wound closed.

The night following the operation the patient had no spasms for the first time for many months. Her improvement was very obvious and has continued ever since.

SUMMARY OF PREVENTION AND TREATMENT

	Prevention	Treatment
Type I—	Not always possible. Hyperextension of definite value	*a.* Primarily conservative *b.* Costo-transversectomy in any severe case likely to become Type II 'continued activity of disease' *c.* Incision of abscess in cervical cases, particularly if there is also marked pharyngeal obstruction
Type II—		
Acute thrombosis of vessels supplying the cord	Not possible	Operation contra-indicated
Concertina collapse	Hyperextension	Operation contra-indicated
Spinal tumour syndrome	Not possible	Laminectomy
Continued activity of disease	Costo-transversectomy while still Type I	Costo-transversectomy
Compression by sequestrum	Hyperextension	Removal of sequestrum should be attempted
Pathological dislocation	Hyperextension	Removal of bony ridge possibly of value
Posterior spinal disease	Not possible	Laminectomy
Type III—	Efficient treatment of the tuberculous spine at every stage	Conservative. In severe cases, possibly laminectomy and graft: removal of tuberculous material or of bony ridge if mechanical compression is present

It has been shown that the efficient treatment of uncomplicated spinal caries is of value in preventing the onset of paraplegia—particularly Type III.

When paraplegia is present the treatment is primarily conservative. In severe Type I cases timely costo-transversectomy is of value, not only in shortening the duration of paralysis, but in preventing the development of Type II paraplegia due to continued activity of the disease.

The less common forms of Type II paraplegia, although very grave conditions, may possibly yield to operative treatment.

In Type III paraplegia we are almost confined to conservative treatment; so far operation has yielded but few successes.

Laminectomy as a routine operation for severe paraplegia is to be condemned; the results are appalling. However, there are a few uncommon but very definite indications for its performance.

The writers of the two preceding papers desire to express their thanks to the many surgeons who gave permission for their cases of Pott's paraplegia to be included in the present series; also to their assistants, and the members of the administrative, nursing, and laboratory staffs whose help has been so valuable.

While it is impossible to mention all by name, they wish to thank particularly the staffs of the Royal National Orthopædic Hospital; Queen Mary's Hospital, Carshalton; St. Luke's Hospital, Lowestoft; Princess Mary's Hospital, Margate; The Royal Sea Bathing Hospital, Margate; The Alexandra Hospital for Hip Disease, Swanley; St. Vincent's Orthopædic Hospital, Pinner; The Shropshire Orthopædic Hospital, Oswestry; and the Hôpital Franco-Américaine, Berck-sur-Mer.

Their thanks are also due to Dr. J. G. Greenfield, Professor H. A. Harris, and to the curators of the museums of the London Medical Schools for permission to investigate museum specimens and for generous assistance in studying material from their own cases.

REFERENCES

[1] ANDRÉ-THOMAS, *Rev. neurol.*, 1924, ii, Feb., 342.
[2] BORCHERS, E., *Münch. med. Woch.*, 1924, lxxi, 652.
[3] CALVÉ, J., *Rev. neurol.*, 1923, xxxix, 711.
[4] CHARCOT, J.-M., *Leçons sur les Mals du Systéme nerveux*, 1880, ii, 88. Paris.
[5] CLARKE, J. JACKSON, *Practitioner*, 1903, lxxi, 407.
[6] COOMBS, CAREY, *Brit. Jour. Surg.*, 1930, xviii, 326.
[7] DÖLLINGER, B., *Die Behandlung der tuberculösen Wirbelentzündung*, 1898, Stuttgart.
[8] ELSBERG, C. A., *Tumours of the Spinal Cord*, 1925, New York.
[9] FICKLER, A., *Deut. Zeits. f. Nervenheilk.*, 1900, xvi, 1.
[10] FINCK, E., *Beitr. z. klin. Chir.*, 1908, lix, 65.
[11] FISCHER, O., *Med. Klinik*, 1919, xv, 1284.
[12] FORESTIER, J., *Le Trou de Conjougaison vertébral et l'Espace epidural.* 1922. Paris.
[13] GIRDLESTONE, G. R., *Brit. Jour. Surg.*, 1931, xix, 121.
[14] GOLDTHWAIT, J. E., *Trans. Amer. Orthop. Assoc.*, 1899, xii, 91.
[15] GOWERS, W., *Diseases of the Nervous System*, 1892, i. London.
[16] GREENFIELD, J. G., and CARMICHAEL, E. A., *The Cerebrospinal Fluid in Clinical Diagnosis*, 1929. London.
[17] HASSIM, G. B., *Histopathology of the Peripheral and Central Nervous Systems*, 1933. London and New York.
[18] KAHLER, O., *Zeits. f. Heilkunde*, 1882, iii, 187.
[19] LANNELONGUE, *Tuberculose vertébrale*, 1888, Paris.
[20] MÉNARD, V., *Etude pratique sur le Mal de Pott*, 1900. Paris.
[21] MICHAUD, "Sur la Méningite et la Myélite dans le Mal vertébral", *Thèse de Paris*, 1871.
[22] MYERS, T. H., *Trans. Amer. Orthop. Assoc.*, 1891, iv, 124.
[23] NUREDDIN, A., *Deut. Zeits. f. Chir.*, 1926, cxcviii, 125.
[24] PAINTER, C. F., and MOORE, G. C., *Amer. Jour. Orthop. Surg.*, 1910, viii, 306.
[25] PELS-LEUSDEN, *Deut. med. Woch.*, 1918, xliv, 143.
[26] POTT, P., *Remarks on that Kind of Palsy of the Lower Limbs which is Frequently Found to Accompany a Curvature of the Spine, and is Supposed to be Caused by it. Together with its Method of Cure.* Tract, London, 1779.
[27] SCHEDE, F., *Münch. med. Woch.*, 1922, lxix, 779.
[28] SCHMAUS, H., *Die Compressions-myelitis bei Karies der Wirbelsäule*, 1890. Wiesbaden.
[29] SHARPE, N., *Amer. Jour. Surg.*, 1923, xxxvii, 142.
[30] SORREL-DEJERINE, *Contribution à l'Étude des Paraplégies pottiques*, 1925. Paris.
[31] SORREL, E., and MME SORREL-DEJERINE. *Tuberculose osseuse et ostéo-articulaire*, 1932. Paris.
[32] SORREL, E., and MME SORREL-DEJERINE. *Rev. neurol.*, 1924, July, 1.
[33] SOUQUES, A., *Ibid.*, 1923, xxxix, 649.
[34] STEINDLER, A., *Diseases and Deformities of the Spine and Thorax*, 1929. London.
[35] STRÜMPELL, A., *Lehrbuch der speciellen Pathologie und Therapie*, 1890, ii, Chap. III.
[36] TILLMANS, H., *Arch. f. klin. Chir.*, 1903, lxix, 134.
[37] VALTANCOLI, G., *Chir. d. Org. di. Mov.*, 1924, viii, 497.
[38] VACCHELLI, S., *Ibid.*, 1 and 244.
[39] VINCENT, C., and DARQUIER, *Rev. neurol.*, 1925, xxxii, 100.
[40] WASSILIEW, M. A., *Arch. f. klin. Chir.*, 1909, lxxxviii, 845.
[41] WHEELER, W. I. de C., *Robert Jones Birthday Volume*, 1928. London.
[42] ZEIGLER, *Lehrbuch d. pathol. Anatom.*, 1885, ii, 627.

SKELETAL LIPOID GRANULOMATOSIS

(*Hand-Schüller-Christian's Disease*)

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INTRODUCTION

It has long been recognized that certain disturbances of lipoid metabolism may constitute a pathology which creates important considerations of diagnosis and treatment, and, while there is an underlying factor common to all the conditions grouped under this heading, there are certain distinctions which justify the adoption of an individual specification. The distinctions are based upon a variety of factors—differences in the biochemical agencies which appear to initiate disease, in the type of tissue affected and the reaction which these tissues display, in the prognosis, and in the response to therapeutic measures. It is upon such grading that a subdivision has been based which includes at present some five varieties of disturbance of lipoid metabolism. They are *Gaucher's syndrome*, in which a lipo-protein of the cerebroside type initiates a morbid anatomy; *Niemann-Pick's syndrome*, associated with a phosphatid lipoid tissue deposit; *Tay-Sachs' syndrome*, probably attributable to a cerebroside protein derangement; *Hand-Schüller-Christian's syndrome*, in which an error in cholesterol metabolism is apparently the factor responsible; and a somewhat *heterogeneous group*, the distinctions of which have not yet been fully investigated, but which may be said to include xanthoma, xanthomyeloma, and xanthelasma, conditions which are attributable to degrees of cholesterolæmia. If an attempt is made to summarize the factors which exert a common and universal influence in what seems to be a concatenation of diseases, it will be found that for certain obscure reasons a state of affairs arises in which an excess of lipoid circulates in the body fluids. A proportion of the excess is taken up by body cells of the histiocyte type, and thereafter a series of cell changes constitutes a pathology which is reflected in various clinical signs, in questions of diagnosis, of prognosis, and of treatment.

The purpose of this paper is to present a study of certain aspects of Hand-Schüller-Christian's syndrome, more particularly in respect of the changes which affect the skeleton; and in so far as it is a lipoid derangement associated with the formation of granulation tissue, and because our interests are more particularly in relation to bone changes, it seems appropriate to use the title of *skeletal lipoid granulomatosis*.

It may seem at first sight that the surgical interests of the subject are by no means evident, but reflection will show that the matter is one of real surgical importance. The criticism may be offered that the condition is so rare that it scarcely justifies a detailed or serious consideration, and the fact that no detailed publication on the subject exists in British literature would seem to support this

view; but the probability is that the belief in its rarity has been exaggerated, for, if a single surgical charge encounters three cases within as many years, the likelihood is that the occurrence of the disease is being overlooked, that the diagnosis is being confused, and that consequently treatment is not always appropriate. For these reasons alone the subject is worthy of consideration, and, if further justification is needed, it may be found in the fact that here is a subject of immense pathological interest, a matter which may well have far-reaching association with problems of tissue reaction and tumour growth.

CASE REPORTS

I feel that it will stress the practical significance of the problem to recount a summary of the clinical histories of four cases which have been under observation in the past few years.

Case 1.—In June, 1928, it was noticed that a female, aged $1\frac{9}{12}$ years, of unexceptional personal and family history, had developed a limp in relation to the left hip. Examination appeared to exclude the possibility of a primary joint lesion, but radiography revealed that the left iliac bone was the site of an extensive rarefactive process. There was no doubt that this lesion, whatever its nature, was the factor responsible for the limp, and, as the diagnosis was uncertain, a biopsy was carried out. Histological investigation revealed the picture of a lipoid granulomatosis showing the characteristic lipoid-containing histiocytes and multinuclear giant cells, features which will be fully discussed when the histopathology is described. I confess, however, that at this stage the histological diagnosis was not established, and it was only in the light of subsequent experience that the real character and significance of the picture were appreciated. In the beginning the appearances were mistaken for those of myeloma, and the questions of prognosis and treatment were oriented accordingly. The further history was that bone changes of a similar nature appeared in relation to the right ilium, the right femur, the bodies of the vertebræ, and the calvaria (*Fig.* 557).

FIG. 557.—*Case* 1. Radiogram illustrating defects in the left parieto-occipital region.

Treatment was instituted by radium packs applied over the affected bones, and by deep radiotherapy, the view being taken that the case was one of multiple tumour formation of the myeloma type. Under this treatment the child gradually improved, recalcification appeared in the rarified zones, and to-day she is in good health, although the areas originally affected still show extensive lacunization. In this instance the error in diagnosis entailed the omission of special lines of investigation, and the case record is therefore imperfect, but, reviewing the condition in the light of fuller knowledge, there appears to be no doubt that the condition was one of lipoid granulomatosis of the cholesterol type.

Case 2.—A male, aged 3 years, was admitted to hospital in November, 1932. It was stated that the first sign of health disorder was noted at the age of 2 years, when the teeth became affected by what was thought to be an extensive infective process. The teeth were extraordinarily soft, crumbling down to the level of the gums, and in several instances were extruded from what appeared to be pockets of inspissated pus. The stature and bony

development were below normal, and the child was described as being irritable and unhappy. These features indicative of bad health were attributed to dental sepsis, though it seemed difficult to understand why such persistent and widespread tooth infection should exist in a child of such tender years. In October, 1932, it was observed that the child was crying a great deal, and from time to time placed his hand over the left ear, as though experiencing pain in that region.

On examination there was found a fluctuating swelling immediately above the left mastoid, and, on the supposition that this was an abscess, the swelling was explored by means of an aspirating needle. A small quantity of faintly blood-stained fluid was removed, and on examination showed the characteristics of cerebrospinal fluid. At this point the child was brought under our observation. He was found to be under weight (38 lb.) and poorly developed, and those who were concerned with the care of him in hospital were soon made aware that he was unusually fractious and irritable. The facies showed slight undue

FIG. 558.—*Case* 2. Antero-posterior radiogram of skull showing defects in left temporal region and left half of the mandible.

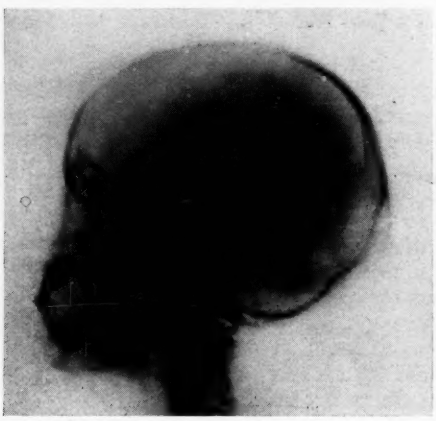

FIG. 559.—*Case* 2. Lateral radiogram of skull showing extensive calvarial defects in occipital and left temporo-sphenoidal region.

projection of both eyes: on examining the skull there was found on the left side an extensive osseous defect involving the greater wing of the sphenoid, the squamous portion of the temporal, and the occipital bone. In this area the pulsation of the underlying brain was palpable and visible. The left half of the mandible was enlarged, as though it were the site of a central cyst or tumour formation. A routine examination of the various systems was carried out; it yielded a negative result except in so far as the urinary system was concerned, in relation to which diabetes insipidus was revealed, 75 oz. of urine being passed in twenty-four hours, with a specific gravity of 1004. The entire skeleton was radiographed, and there was found an extensive lacuna in the left spheno-temporo-occipital region with smaller lacunæ in the frontal, the right parietal, and the occipital regions (*Figs.* 558, 559). The swelling of the left half of the mandible was shown to be due to a central cyst expanding the cortex.

I confess we were at a loss to know the explanation of the various findings, or to trace the thread by which they might be linked up and associated, and it was at this stage that we were fortunate enough to enlist the interest of Mr. David M. Greig, the Curator of the Royal College of Surgeons Museum in Edinburgh. He taught us wisdom on many points, and it was he who suggested that the case under observation might be an example of the Schüller-Christian syndrome, or, as he preferred to designate it, skeletal lipoid granulomatosis.

Further investigation was accordingly made. The cell picture of the blood was normal; the blood calcium was slightly raised (11·6 mgrm. per cent), the calcium output of the urine

was normal; the blood-cholesterol was raised to 267 mgrm. per cent. One of the skull lacunæ was explored; there was found to be complete disappearance of the ossified tissue, the gap being occupied by a quantity of yellowish rubber-like material which was closely adherent to the underlying dura mater. This tissue on being submitted to microscopical examination showed the characteristic appearance of a granulomatous disturbance, while the presence of histiocytes, foam cells, and multinucleated giant cells made it clear that the lesion was of the Schüller-Christian type.

The fate of this child was extremely unfortunate. After a course of deep radiotherapy, from which he derived considerable benefit, he returned home, to succumb to an attack of post-influenzal pneumonia in February, 1933.

Case 3.—A male, aged 4 years, born in India, of whose early medical history no more need be said than that he was described as an underdeveloped and somewhat irritable baby. He had the reputation of being a remarkably thirsty little creature, and it may be allowed that he deserved such a description when I say that he sometimes consumed three quarts of water in twenty-four hours. In December, 1932, a swelling the size of a cherry was discovered over the centre of the occipital bone. It was believed to be a sebaceous cyst, but, as it was increasing in size, its removal was decided upon, and this was carried out in January, 1933. Operation revealed that the diagnosis was erroneous; the tumour was a solid one, and not only so but it was associated with disappearance of the underlying bone, while it showed evidence of involving the dura mater. The appearance suggested a malignant condition, and, when the tissue was submitted to microscopical examination, the worst fears appeared to be confirmed, for the pathologist reported that the tumour was a sarcoma. About this time what seemed to be irrefutable confirmation of the malignant character of the lesion was demonstrated when a tumour of the left clavicle, assumed to be a secondary deposit, made its appearance.

In view of the gravity of the prognosis the parents were advised to return to this country as soon as possible, and the child came under our care in March, 1933. The appearances by that time were in many respects characteristic. The child was nervous and irritable; he was small for his age and considerably under weight (his weight at 4 years being 22 lb. and his height 2 ft. 8½ in.); he was particularly resentful of any attempt at examination; his eyes showed a slight degree of exophthalmos; the scalp veins were unduly prominent, particularly when the child strained or cried. On handling the skull there was found to be an extensive defect of the calvaria in the occipital region, the area measuring 4 cm. by 5 cm.; there was a smaller lacuna in the centre of the left parietal bone. The left half of the mandible was enlarged, while the medial half of the left clavicle showed a fusiform enlargement the size of a bantam's egg. No abnormality could be detected in the rest of the osseous system. The teeth were extremely unhealthy, many being worn to the gum level, while the two anterior to the molar teeth in the left half of the mandible were obviously loose, and it was noticed that when they were 'rocked' by the finger a small quantity of yellowish inspissated material resembling pus appeared around the edges. The posterior chain of the deep cervical lymph nodes on both sides of the neck was enlarged, each gland being about the size of a hazel nut; there was no pain associated with the enlargement, and there was no evidence of periadenitis. The respiratory, circulatory, and alimentary systems appeared to be normal. Investigation of the urinary system yielded some interesting results. There was a pronounced polyuria, an average of 90 oz. being passed in twenty-four hours, and in relation to this degree of polyuria there was a corresponding degree of polydypsia, over 100 oz. of fluid being consumed in the same period of time. The blood showed a normal picture so far as red and white cell counts and hæmoglobin readings were concerned, but a biochemical investigation revealed a definite increase in the lipoid content, the readings being as follows: Total cholesterol 236 mgrm. per cent, total fatty acid 287 mgrm. per cent, lecithin 274 mgrm. per cent. This result revealed a moderate degree of cholesterolæmia if one accepts the normal cholesterol content of the blood as 100 to 150 mgrm. per cent.

A radiographic investigation of the entire skeleton was carried out. The occipital and right parietal bones were the sites of extensive lacunæ, the edges of which showed a characteristic irregular map-like outline (*Fig.* 560); the centre of the body of the left half of the mandible was occupied by a cyst-like formation, and it was interesting to notice that the apices of certain of the teeth appeared to lie within the confines of this cavity. The medial half of the left clavicle contained a central cyst-like deposit, which was causing expansion

of the cortical bone and absorption of the cancellous interior. The remainder of the osseous system appeared to be healthy.

At this stage an opportunity was afforded of examining sections of the skull tumour which had been removed in India in January, 1933, and it was found that the microscopical picture was characteristic of a lipoid granulomatosis of the Schüller-Christian type.

FIG. 560.—*Case* 3. Lateral radiogram of skull showing extensive calvarial defects.

The diagnosis of the condition appeared to be fully established, and under a scheme of treatment which included deep radiotherapy and pituitrin injections the child improved. He is at the present date in reasonably good health, but in the progress there have been two episodes which are significant of the disease and of the way in which it may be associated with problems in local diagnosis.

In October, 1933, pain was complained of in the left half of the mandible, and on the supposition that there was a tooth infection, the left canine of the first dentition was removed. It was found that the buried portion of the tooth was covered with a yellow caseous-like substance, while a quantity of a similar material (which as a matter of fact was mistaken for pus) escaped from a cavity in the jaw with which the apices of the tooth had been communicating. A subsequent analysis of this substance showed that it was largely composed of cholesterol. (*See Fig.* 565.)

In November, 1933, the child developed the signs and symptoms of acute right mastoid infection. Operation was carried out, when there was found to be an extensive infiltration of the mastoid sinus and the squamous portion of the temporal bone by a deposit similar to that which had been encountered in relation to the teeth. The appearances suggested inspissated pus, and no doubt there was a certain element of infection, but analysis showed that the deposit was largely composed of cholesterol.

Case 4.—I am permitted to quote this case by the courtesy of Dr. Leila Hawksley, Pathologist to the Cancer Hospital, Fulham Road, London, and Professor Woodburn Morison. I take the opportunity of expressing my indebtedness to them for allowing me to study the case history, and examine various radiograms and pathological material illustrative of the case.

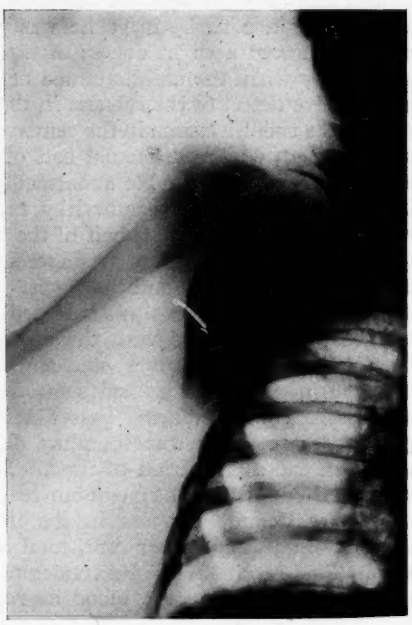

FIG. 561.—*Case* 4. Radiogram showing the tumour of the right scapula. (*By permission of Professor Woodburn Morison.*)

A female, aged 8 months, was brought to the Out-patient Department of the hospital in January, 1932, with the history that a month previously it was noticed that she was not using the right arm freely. Examination showed that there was a swelling involving the infraspinous area of the right scapula (*Fig.* 561); it was ovate, some 60 mm. in length, and on radiography it was found to be associated with an expansion and erosion of the bone in an area immediately inferior to the glenoid cavity. No other bones were affected, the various systems appeared to be healthy, there were no blood changes, no polyuria, and no disturbance of the eyes.

On Jan. 14 the swelling was explored, and a portion removed for microscopical examination. The histopathology illustrated appearances suggestive of a lipoid granulomatosis, that is to say, lipoid-containing cells of the histiocyte type and multinuclear giant cells. The tumour was treated by radium applied interstitially, a dosage of 2,568 radium-hours being given.

In July, 1932, the child was re-examined. The swelling of the right scapula persisted, and there was found a small tumour arising at the centre of the left parietal bone, while the calvaria in an area immediately underlying the tumour had disappeared. The child was kept under observation, and by November, 1932, further bone deposits appeared in the skull, the left scapula, the right ilium (*Fig.* 562), and the first rib, the skull deposits being associated with palpable areas of tumour formation. Meantime the child's general health showed deterioration, but there was no evidence of any visceral or blood change; the Wassermann reaction was negative. Further bone deposits now made their appearance in the skull (*Fig.* 563), the left mandible, the left clavicle, and the right ilium. There is no record that polyuria existed.

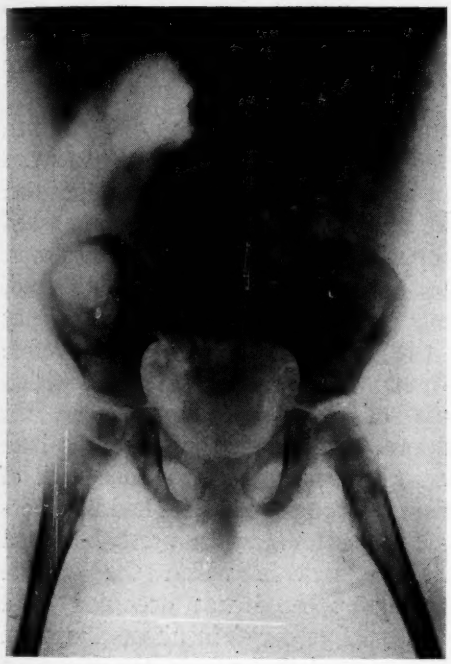

FIG. 562.—*Case* 4. Radiogram of the pelvis illustrating the defect in the right ilium. Note also the deposit in the neck of the left femur. (*By permission of Professor Woodburn Morison.*)

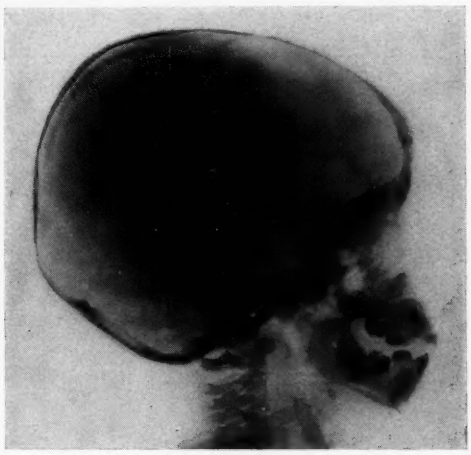

FIG. 563.—*Case* 4. Radiogram of the skull. The chart-like outline of the skull defects is well shown. (*By permission of Professor Woodburn Morison.*)

Deep radiotherapy was begun in November, 1932, and from that time improvement became evident. The last report indicated that the skull tumours had disappeared, and that the lacunæ were being replaced by new bone. The other bone tumours also showed signs of improvement, and the child appeared to be in good general health and developing normally.

These are the presentations of four cases which show certain features in common—deposits, chiefly in the membrane bones, of such a nature as to be mistaken for malignant tumour formation, and illustrating a pathology which, while it bears resemblance to primary tumour formation, appears to have its explanation in a stimulus of reticulo-endothelial tissues by a chemical substance of a lipoid character. The cases may be regarded as illustrating the disease in the early stages of its progress, and, as we shall see, a later and more extensive pathology implies a wider and more varied clinical history.

THE HISTORICAL ASPECT

Before discussing the various aspects of the condition which these cases illustrate, I propose to present a short historical account of the disease, for it is interesting to recall the phases and stages by which one's knowledge of the disturbance has advanced.

Priority of recognition is generally ascribed to Alfred Hand,[1] who published in 1893 a case showing a trilogy of features—calvarial osseous defects, exophthalmos, and polyuria. The condition was undoubtedly one of lipoid granulomatosis, but Hand, misled by the caseous-like appearance of the granulation deposit, attributed the changes to tuberculosis. Literature, however, contains an earlier record, and, though the story is incomplete and the diagnosis was unsuspected, there is abundant interest in the facts. I am indebted to Mr. David M. Greig for bringing this case to my notice. The details are as follows :—

In 1865 Thomas Smith,[2] of London, examined a child 4½ years old who had developed a swelling over the occipital bone. The swelling was described as soft and fluctuant, and on the supposition that it was an abscess, Smith was asked to operate upon it. He found, however, that, though fluctuant, it also pulsated, while there was an evident deficiency in the underlying skull. He therefore refused to incise the swelling. Other areas of deficiency appeared in the calvaria, and, when some months later the child succumbed to an attack of whooping-cough and Smith had an opportunity of examining the parts, he found that the area in which the original swelling appeared was occupied by a quantity of yellowish inspissated material which he described as "a dried-up abscess". When the appearance of the calvaria as figured in Smith's paper is recalled (*Fig.* 564), there can be little doubt that the case was an example of lipoid granulomatosis. The record is, of course, incomplete, and there are many details about which, in the light of present knowledge, we would like information, but the evidence which exists suggests that here was an example of the disease with which this paper is concerned.

FIG. 564.—The skull of Thomas Smith's case, showing the characteristic calvarial defects. (Reproduced from Smith's original article, published in the *Transactions of the Pathological Society of London*, 1865, Vol. xvi.)

I have already alluded to Hand's case. He published the details in *The Archives of Pediatrics*, 1893, under the heading "Polyuria and Tuberculosis", and, according to his description, what he regarded as tuberculous deposits in the calvaria and skull base were associated with the symptom of polyuria. Everything points to its having been a case of lipoid granulomatosis manifesting the signs of pituitary involvement.

In September, 1905, Thomas Kay[3] presented before the Medical Society of Pennsylvania a boy 7 years of age who showed a clinical picture presenting exophthalmos, polyuria, and extensive defects of the calvaria. Kay expressed the opinion that in this case a lesion existed in or about the floor of the fourth ventricle, but he appears to have made no attempt to explain why a lesion in this situation should be associated with osteoporosis of the bones of the cranial vault.

In 1915 Artur Schüller[4] described three cases of varying symptomatology, each of which showed defects with irregular outline affecting the membrane bones: a lad of 16 years presented the picture of infantilism, exophthalmos, and the adiposogenital syndrome; a female child of 4 years evidenced stunted growth, exophthalmos, and diabetes insipidus; while in the third case (an adult) there was an infiltration of the skin with yellowish nodules. Schüller did not suggest any common etiological factor in relation to these cases; they were presented in virtue of the radiological interest of the skull defects, but in connection with the girl he ventured an opinion that the skull defects and the diabetes insipidus were caused by a pituitary disturbance, and that the exophthalmos was the result of the pressure of the brain on the orbital contents, being made possible by the large defects in the anterior fossa of the skull, and more particularly in the orbital plates.

Under the heading "Defects in the Membranous Bones, Exophthalmos and Diabetes Insipidus", Henry Christian[5] published in the *Osler Memorial Volume* of 1919 the report of the case of a girl 5 years old who had been under his care in the Peter Bent Brigham Hospital in Boston. The case presented a typical picture of the symptom complex which the heading implies. In a summary of the detailed case report Christian said, "Diabetes insipidus suggests that the symptom complex is due to a disturbance of pituitary function", and he pointed out that the polyuria was controlled by intramuscular injection of pituitrin. Christian reprinted his original article in the *Medical Clinics of North America*,[6] and subsequent to his publication the condition which he described came to be known as Schüller-Christian's disease.

In 1921 Hand[7] encountered a case somewhat similar to that which he had reported in 1893. In this instance a child 3 years old was found to be suffering from exophthalmos, a corneal opacity in each eye (probably the result of exposure), polyuria to the extent of passing 114 oz. of urine daily, and an area of softening in the centre of the right parietal bone. Hand's clinical diagnosis was one of tuberculosis, as it had been in 1893, but on this occasion the child succumbed to an attack of bronchopneumonia, and, as a result of a subsequent autopsy, he advanced the theory that "the primary process might be neoplastic, benign, and myxomatous in character, affecting for some unknown reason the membranous bones and producing exophthalmos and polyuria secondarily by pressure." The pathological report contains a significant statement to the effect that the defect in the centre of the right parietal bone was found to be occupied by a soft yellow substance not unlike caseating material.

The next record with a bearing on the problem is by Thompson, Keegan, and Dunn.[8] A boy of 9 years of age presented the classical triplication of features —exophthalmos, calvarial defects, and polyuria. The child died, death being attributed to cardiac failure as the result of extensive pulmonary fibrosis. The authors give a detailed account of the autopsy findings, and in describing the condition of the skull they say, "In the centre of the largest membranous areas

(i.e., the calvarial defect) there was yellowish fibrous tissue, and the inner surface of the dura mater was mottled by a yellowish tissue. Microscopical examination of the area showed a lining layer of large oval cells with sharp borders and clear, slightly granular cytoplasm, and small compact centrally placed nuclei. The cytoplasm contained a variable amount of lipoid material in finely divided form." In estimating the cause of the disease, the authors concluded that an infection of some kind existed rather than a metabolic endocrine disturbance, but from the historical point of view the main interest lies in the fact that *this was the first instance in which the existence of lipoid in the lesion was described.*

What appear to have been characteristic cases were reported by Grosh and Stifel[9] in 1923 (female, 7 years old) and Denzer[10] in 1926 (boy, 4½ years), but these authors had no suggestions to offer regarding the problem of etiology.

Artur Schüller,[11] whose contribution to the literature of the disease has already been mentioned, communicated a second paper at the International Congress of Radiology in London in 1925 on what he called " Dysostosis Hypophysaria." He reviewed the cases which had hitherto been reported, and the summary of his conclusions was as follows :—

> There exists a peculiar type of defect of the skull characterized by the multiplicity, the great size, and the sharp outlines of the defects, localized as well on the cranium as on the base of the skull, combined with defects in the pelvis, exophthalmos, and with some of the symptoms generally diagnosed as due to pituitary dysfunction, namely dwarfism and diabetes insipidus. I propose for this symptom complex the name of Dysostosis Hypophysaria.

It is evident from these remarks that Schüller maintained the view which he expressed ten years before, that the disorder was primarily of pituitary origin.

In 1926 Kyrklund[12] published the case records and the autopsy findings of a 14-year-old-girl who four years before had developed polydypsia and polyuria, and who ultimately succumbed to cardiac weakness. He records characteristic findings in the calvaria and skull base. The hypophysis did not show pathological findings, but there were brownish-yellow deposits in the brain-stem behind the hypophysis. He mentions that the lungs showed extensive changes. Kyrklund's final conclusion was that the deposits were of a sarcomatous nature, and that the changes in the brain-stem behind the hypophysis were responsible for the diabetes insipidus.

In 1927 a case, the clinical findings of which were reported by W. R. Stowe,[13] came to autopsy, and D. S. Milne has given us an interesting and important account of the pathological findings. I shall quote a portion of his report.

> On reflecting the scalp a number of swellings resembling putty in appearance but of fairly firm consistency were observed. These areas were extensions through small perforations in the skull from the dura, which was extensively infiltrated. The brain was normal; a compact yellow mass about the size of a walnut rose up out of the sella turcica, displacing the hypophysis, but without eroding or distorting the clinoid processes. Another mass projected back from the right wing of the sphenoid, which was extended to the ethmoid and both orbits.

Continuing with a report of the histology of the abnormal tissue, Milne states :—

> The striking yellow mass [was] composed of spindle cells with clear spaces filled with granular yellow substance. . . . Scattered throughout the tumour were giant cells with rosette nucleus surrounded by a granular zone. The nodules from the dura showed the same changes. The entire pituitary was invaded by the yellow growth.

The exact nature of the error appears to have been the source of considerable difference of opinion. Milne came to the conclusion that the condition represented a tumour arising in the hypophysis; Dr. Lynch suggested that it might be a chordoma, though as an alternative he put forward the suggestion that the tissue showed many of the characteristics of a xanthoma, and this may be said to be the first hint as to the true nature of the condition.

In 1928 R. S. Rowland,[14] of Detroit, published an article in the *Archives of Internal Medicine* which supported Lynch's view of the origin of the disease. The title of the article is "Xanthomatosis and the Reticulo-endothelial System", and in a more elaborate title the contribution is described as "Co-relation of an Unidentified Group of Cases described as Defects in Membranous Bones, Exophthalmos, and Diabetes Insipidus (Christian's Syndrome)". Rowland reported two cases illustrative of the syndrome, one of which came to autopsy, the subjects being both male children, aged 5 years and 3 years and 4 months respectively. Rowland's conclusions on the nature of the condition amounted to this—that he regarded the cases as representing a form of xanthoma in which portions of the reticulo-endothelial system became the site of excessive lipoid storage, with resulting hyperplasia of lipoid-containing cells. In his own words: "One is not dealing with true neoplasm, but with hyperplastic new formation, i.e., lipoid storage tumours. The formation of these nodules is a compensatory act on the part of the body in its attempt to rid the blood of an excess of lipoid which cannot be properly excreted."

In 1929 Hausman and Bromberg,[15] in reporting a case which had been under treatment in the Bellevue Hospital, New York, on account of pronounced exophthalmos, described the deposits which occur in the region of the hypophysis, and drew attention to the fact that the changes are often relatively widespread, affecting not only the pituitary but also the stalk and the tuber cinereum, and in this connection they raised the interesting possibility that the para-infundibular unit may in some way exercise an atrophic influence upon bone, so that a pathological disturbance of the area may be the factor responsible for the defects in the skull bones.

Since 1931 there have been a number of additional references in literature, but in this survey I have been satisfied with outlining what may be called the milestones in the recognition of the disease, and, for those who may be particularly interested, a moderately complete bibliography is appended (p. 824).

A CONCEPTION OF THE DISEASE

As a result of clinical observation and a certain amount of experimental research, a more or less precise conception of what the disease implies has now been arrived at, and, though there are many points which are still in dispute, we have a fair appreciation of its characters.

Lipoid granulomatosis (and I prefer to employ this collective terminology) is a process by which lipoid substances are deposited in selected tissues, with the result that the areas so affected become the sites of granulation tissue formation bearing a close resemblance to malignant tumour, and, in virtue of a destructive influence upon certain organs and tissues, a characteristic syndrome of clinical features is produced. As I have already indicated, there is variation in the type of lipoid, and in association with these variations in type different groups of tissues are affected and different sites of deposit selected. It is upon this basis

that we encounter such clinical conditions as Niemann-Pick's disease, in which a lipoid of the lecithin or phosphatid type is deposited in the lymph-vascular and hæmopoietic tissue of the liver, the spleen, and the bone-marrow; Gaucher's disease, in which a lipo-protein of the cerebroside type is deposited in a similar group of tissues; xanthomatosis, in which a cholesterol lipoid appears in relation to epithelium; Tay-Sachs' disease, the essential feature of which is an accumulation of a phosphatid lecithin lipoid in the tissues of the central nervous system; Schüller-Christian's disease, the condition with which this paper is more particularly concerned; and probably others not yet investigated or classified.

In this relatively wide field we are concerned with one variety of the disturbance. It is that which is known to the medical profession as Schüller-Christian's syndrome, and its basis is a deposit of cholesterol lipoid in relation to reticulo-endothelial tissue, and more particularly the reticulo-endothelial tissue of membrane bones, serous surfaces, and vascular areas.

In order to present a reasoned appreciation of the disease, I propose to summarize certain of the more important facts regarding the two elements which govern and control its development, these elements being the reticulo-endothelial tissues and the cholesterol lipoid.

The Reticulo-endothelial Tissues.—The recognition of the tissues which comprise the reticulo-endothelial system may be said to depend upon two properties—the morphological peculiarities of the cells as revealed in staining reaction, and the function which the cells appear to provide. All that need be said in respect of the former is that cells in this group show a vital staining reaction when submitted to such a stain as trypan blue; that is to say, they possess the property of absorbing and retaining the granules of the dye within the substance of the cytoplasm, and there is the further fact that the physiological significance of the cell appears to depend upon the degree to which the stain granules are accepted and retained. In describing the distribution of reticulo-endothelial tissues the term 'ubiquitous' might well be applied, for the system includes: (1) The endothelium which lines blood- and lymph-vessels; (2) The adult connective-tissue cell or fibrocyte; (3) The cohesive reticulum cell of splenic and lymphoid tissue; (4) The reticulum and endothelial cells which lie in relation to the sinusoids of the spleen, the lymph nodes, the liver, the bone-marrow, the suprarenal cortex, and the hypophysis; (5) The large phagocytic and motile cells of connective tissues (macrophages or histiocytes); (6) The blood monocytes. In this widespread distribution, if we estimate functional importance upon the basis of such a morphological standard as staining reaction, the evidence would seem to indicate that it is the fourth group, the reticulum and endothelial cells lining the sinusoids of the spleen, liver, lymph nodes, bone-marrow, etc., which play the most active part in the physiological activity of the series.

The functions of the system are as yet imperfectly understood, but it seems that cell reproduction, phagocytosis, and certain metabolic processes are probably its most important activities. With the first of these we are not for the moment concerned beyond indicating that the process of cell reproduction or metamorphosis tends to focus itself upon the formation of such active phagocytic elements as large monocytes, histiocytes, and macrophages of the polyblast type. But, for reasons which will presently become apparent, we have special interest in the phagocytic activity of the system. It is evidently a remarkably efficient arrangement, for its

activities are apparent on a variety of tissues—on other cells in the form of red and white blood-corpuscles, muscle fibres, and damaged cells of any type, on such foreign materials as the granules of stains and dyes, on bacteria, and on such metabolic products as hæmoglobin and lipoid. The majority of these various phagocytic activities must be regarded as reactive, but when exercised in relation to products of metabolism may be described as functional, for it is a physiological reaction, a necessary attribute of health. It may seem a misnomer to speak of this last rôle as phagocytic, but in so far as it is a cell inclusion it is such, only in this instance it extends beyond a simple inclusion and destruction, for the inclusion is but a stage in a further metabolic change. Thus we find that hæmoglobin is phagocytosed in preparation for its conversion into bilirubin, while the lipoids, and more especially cholesterol, are phagocytosed for purposes of storage, and no doubt for subsequent release when such is demanded.

In summary, therefore, we may say that the reticulo-endothelial system is a tissue arrangement of widespread and general distribution, that it finds its most efficient arrangements in relation to the blood-sinuses of certain viscera, to bone-marrow, and to serous surfaces, and that among its most striking properties are its phagocytic abilities in conjunction with its power of including, storing, and metabolizing such substances as hæmoglobin and cholesterol.

The Cholesterol Lipoid.—The evidence we possess indicates that an excess of cholesterol in certain body tissues is the primary factor in the production of the clinical syndrome of lipoid granulomatosis (Schüller-Christian's disease), and to make this discussion sufficiently inclusive it is well that we should examine certain of its more important properties and functions. It is one of the large group of fatty substances which, in conjunction with proteins, occurs in the protoplasm of body cells, and to this group the inclusive term 'lipoid' is applied. This word was first introduced in relation to the activity of certain narcotic drugs, for these substances are soluble in lipoids, and, in view of the relatively large percentage of lipoids which exist in nerve-cells, it is believed that it is the alteration in the physical condition of nerve-cells resulting from the solution of the drug in the lipoid which results in narcosis.

According to Cameron,[16] cholesterol is one of the group of simple lipoids, in contrast with the compound or complex lipoids. It may exist in pure form, in which event it is an unsaturated monohydric alcohol, or it may occur as an ester, being combined with a fatty acid as a palmitate, an oleate, or a stearate. Cholesterol and its esters are present in nerve-tissue, in red blood-corpuscles, in bile, in lymphoid tissue, in the spleen, in the adrenal cortex, and in conjunction with fats throughout the body generally. Its functions are not clearly understood, but, according to Wile[17] and his co-workers, cholesterol exerts a protective action, by which body cells are preserved against the influence of such toxins as are liable to enter the body through the avenue of the alimentary canal or of the skin. It exists in the envelope of red blood-corpuscles in order to prevent hæmolysis and destruction of the blood-cells by organisms or their toxins. Under certain pathological conditions it is deposited in appreciable quantities, as when it is encountered in gall-stones, in the atheromatous patches of arteries, in the walls of tuberculous foci, and in carcinomatous tissues.

In speaking of the question of the formation and the metabolism of cholesterol, Cameron, whose views have been already quoted, says :—

While normal diet contains cholesterol, or sterols easily convertible into cholesterol, evidence is accumulating that the animal organism can synthetize the compound as need arises; for example, the developing chick shows an increase of cholesterol which can only have been formed by synthesis; a newly-born dog fed on a diet poor in cholesterol for four weeks shows at the end of that time an increased cholesterol twenty times the amount administered in the food.

That cholesterol is subject to a certain amount of metabolic change in the body tissues seems to be suggested by the fact that intestinal juice, pancreatic juice, bile, and blood contain an enzyme which will hydrolyse cholesterol esters, while pancreatic juice contains an enzyme which will synthetize the esters from their constituents. During its passage through the intestine a considerable portion of the cholesterol is reduced to coprosterol ($C_{27}H_{17}.OH$) by bacterial action.

Such is a summary of the physiology of the two elements, derangements of which constitute the essential pathology of lipoid granulomatosis.

The Constancy and the Origin of the Hypercholesterolæmia.—Let us now consider the problem from a local standpoint. When a lipoid granulomatosis error arises, one finding is constant—it is that of an excess of lipoid in the body tissues, and more particularly in the blood and lymph fluids. It has been reported by Christian, Rowland, Sosman, and others, and of the three cases which came under personal observation the two which were investigated from this point of view showed a definite cholesterolæmia. There seems to be no doubt that this factor is the original and essential feature underlying the local manifestation, and such being the case, it may be that an attempt on the part of certain body tissues to eliminate a portion of the excess results in the individual pathology. It is therefore reasonable to inquire as to the factors which control lipoid metabolism, for it is by an understanding of these that we are likely to arrive at a knowledge of the conditions which are responsible for the increase which characterizes the disease.

Raab[18] claims that the process is under the control of the autonomic nervous system, initiated in the pituitary gland, whence impulses are relayed by the autonomic centre in the floor of the third ventricle and the hypothalamus to the sympathetic fibres, which in turn act as a mobilizer of stored lipoids, at the same time promoting the destruction of any excess in the liver. This theory is of peculiar interest in relation to the subject under discussion, for there is a stage in the progress of the disease in which there is evident derangement of the pituitary gland and the hypothalamus, but, as we shall see, the pituitary-hypothalamic involvement is a relatively late stage in the pathological sequence, so that any influence which such changes may exercise can only be secondary in their effect. While there is no evidence of a primary pituitary disturbance, it may be, if Raab's views are correct, that the changes which are ultimately wrought upon the gland in lipoid granulomatosis are a factor in maintaining a hypercholesterolæmia.

I believe I am correct when I say that, according to the views of the majority of biochemists, the liver and the lungs are the visceral areas in which the cholesterol balance is maintained, and in support of this contention the significant fact is quoted that blood from the portal vein contains a larger proportion of cholesterol than that from the hepatic veins, while similarly the percentage in the pulmonary artery exceeds that found in the pulmonary veins. If we apply the truth of this finding (Cameron) we may argue that the factor responsible for the occurrence of hypercholesterolæmia is a disturbance of the tissue arrangements in the liver and

in the lungs, so that either an excess of lipoid is produced or the process of destruction which maintains the balance is interfered with. At first sight it might seem that an increased ingestion of lipoids offers the most likely explanation of a body excess. A great deal of experimental work has been done on this aspect of the question; the results are by no means conclusive, but the fact emerges that, as soon as an increased intake is established, that which constitutes an excess is dealt with by cells of the reticulo-endothelial system. So far as I am aware, the position is that, while an increase of the lipoid ingestion may result in a temporary excess of lipoid in the body fluids, the balance is quickly adjusted through the medium of the reticulo-endothelial system, and it is impossible to institute a hypercholesterolæmia for any length of time. Experimental proofs of this truth are numerous. Anitschkow and Chalatow[19] fed rabbits on a diet rich in cholesterol; after some time the histiocytes of the spleen and lymph nodes were found to be loaded with the lipoid, but no appreciable increase was apparent in the blood or lymph fluid.

Working on the same lines, Yuasa[20] obtained very similar results. He employed omnivora in his experiments, because he felt that in this instance cholesterol would not constitute a foreign article of diet, as it might if herbivora were used.

Fontana[21] and his colleagues fed rabbits on large amounts of viosterol. They confirmed the results of previous observers in regard to the response shown by reticulo-endothelial cells, and in more than one instance the cell reaction in the histiocytes of the spleen was so pronounced as to result in a definite enlargement of that organ with a histology very similar to that which is encountered in the lipoid granulomatosis of Niemann-Pick's disease. The experiments do not record any coincident increase of lipoid in the circulating body fluids.

These various observations have brought out one feature of much practical significance, which is fully elaborated by Chalatow[22] in his most recent paper, and it is this—that when lipoid deposits are produced in the body tissue by such experimental means as feeding animals on an excess lipoid diet, the deposits appear to arise simultaneously and suddenly in what might be described as a shower (*Springartige*), an evidence which suggests that the body possesses a threshold to lipoids, as it does to other substances. If the threshold is passed, the substance becomes foreign to the body and is dealt with by a widespread process of inclusion and subsequent destruction.

If we sum up the experimental evidence in so far as it helps to illuminate the problem of hypercholesterolæmia, the findings indicate that the excess of lipoid in circulation probably depends upon a failure on the part of the liver and lung tissue to maintain the body lipoid at a normal level; as a result the reticulo-endothelial tissues attempt to eliminate the excess by a process of absorption and deposit, and, according to Chalatow, the deposit is a multiple and simultaneous one accomplished as soon as the physiological threshold is exceeded.

THE ORIGIN OF LIPOID GRANULOMATOSIS

In attempting to offer an explanation of the process which results in the deposit of localized collections of cholesterol so as to constitute the clinical condition of lipoid granulomatosis (Schüller-Christian's disease) there must be a considerable element of hypothesis. Bearing this in mind, it is suggested that

the following is the outline of events, as we suppose they developed in the cases which have been described.

The initial change is one of increased lipoid (cholesterol) content in the circulating body fluids. The possible explanations of this have been discussed, and, though there is no certainty as to the exact nature of the elements which initiate the disorder, there is reason to suppose that it originates in some change (possibly prenatal) occurring in the areas which control lipoid (cholesterol) metabolism—the liver and the lungs. As a result of the lipoid increase, and in an attempt to adjust the balance, the reticulo-endothelial tissues of certain body areas proceed to concern themselves with absorbing and depositing the substance so that accumulation occurs in individual areas. This phase is not accomplished without considerable cell reaction among the tissues concerned, and the changes which ensue may be said to constitute the characteristic pathology of the lesion.

MORBID ANATOMY AND HISTOPATHOLOGY

The morbid anatomy of the fully established disease may be said to consist in the deposit in certain areas of a granulation tissue of characteristic appearance. The collections are firm in consistency and putty-like in appearance; in certain instances, apparently where the lesion has been longest in existence, the tissue may assume a yellowish coloration.

Deposits of this nature may be encountered in a variety of situations, and from our own observation, together with a study of the literature, the following sites have been recognized: the sub-calvarial dura, whence it extends into the diploë,

FIG. 565.—*Case* 3. Two teeth from the mandible. Note the lipoid deposit which covers the roots and the bodies.

probably along the line of an emissary vein, and ultimately destroys large areas of the bone; the dura of the skull base, from which area there is extension into the sella turcica, and through the superior orbital fissure into the orbit; the cancellous centre of the lower jaw, in which event it possibly is an extension from the vascular tissues at the apices of the teeth (*Fig.* 565); the mastoid air-sinus; the clavicle; the pelvic bones; the ribs; the vertebræ and the long bones; the cervical lymphatic nodes; the pleuræ and the lungs; the liver, where the change has been noted in relation to Kupffer's cells; the macula; and the left cerebellar hemisphere (Cushing). Microscopic lesions suggestive of the pathology have been described in the stomach wall (Davidson[23]), but nothing abnormal has been noted in the suprarenals, the pancreas, or the thyroid.

It is evident from this summary that the distribution of the deposits is widespread, but it is also apparent that there are certain sites of selection, and that these are the mesothelial areas of the basal and calvarial dura and the pleura. Why this selection should be exercised is difficult to explain, unless it be that the reticulo-endothelial tissues of these areas possess some special function in dealing with lipoid excess. One might anticipate that the spleen would form such an area, but, as far as I am aware, no evidence of its selection has been encountered.

The various cytological changes which underlie the development of the granulation tissue have been made the subject of detailed observation, and the phases have been described by Sosman[24] and others. The process seems to

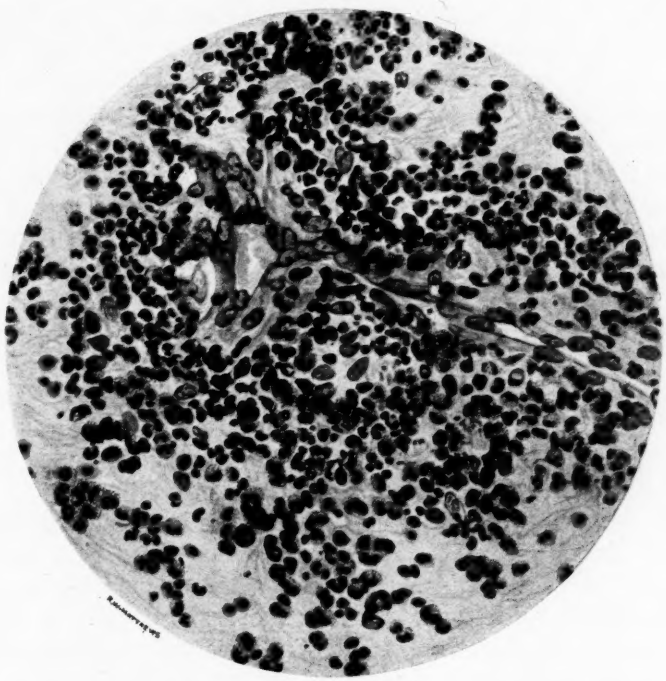

FIG. 566.—Drawing of a section showing an early phase of the pathology. The endothelial cells lining a capillary have become swollen and detached from the vessel wall. A certain number have begun to migrate into the surrounding tissues. Many of the cells show a commencing lipoid accumulation in the cytoplasm. (× 300.)

be as follows. Recognizing the essential initial feature that the body fluids, and the blood in particular, contain an excess of cholesterol content, in one area characterized by a profuse capillary circulation there is a blockage of a capillary lumen as a result of swelling and proliferation of its lining endothelium (*Fig.* 566). It is not clear why this change should occur; it has been suggested that it is due to local injury, but on the other hand it may be that it is the result of a cholesterol concentration acting as an irritant. Whatever the cause, the endothelial swelling and proliferation are such as to constitute a local barrier to the capillary circulation, and from this centre the subsequent pathological changes spread and develop. That an irritant process of some kind is at work is suggested by the succeeding phase, for around the obstructed area an intense concentration of lymphocytes and

eosinophilic leucocytes becomes evident (*Fig.* 567). In the meantime the capillary area loses all trace of its tubular structure, and the point at which the original changes appeared is occupied by a number of oval and fusiform cells which in respect of their staining reaction show evidences of their vascular origin. Scattered among the mononuclear cells are giant cells of a multinuclear type which have obviously arisen by a syncytial process. Lipoid deposits now become evident in the cell cytoplasm (*Fig.* 568). The lipoid is first recognized in the mononuclear cell which has been derived from the vascular endothelium, and its appearance synchronizes with an interesting development, for the lipoid-containing cells proceed to congregate in syncytial-like masses so as to constitute the so-called multinuclear giant cells of the lesion (*Fig.* 569). A stage is eventually reached in which the lipoid-laden cells either discharge their contents or lose their definition as cells, and become amalgamated with a mass of lipoid and cellular débris. In so far as the cell reaction is concerned, interesting individual facts emerge, two of which are that the lipoid, when it enters the cell, collects in the perinuclear area, and that cells so affected cease to show any sign of nuclear division (*Fig.* 570). The latter is a remarkable feature; it has been pointed out by several observers, and it cannot fail to arouse interest that a tissue which shows wide variation in cell structure and type should be characterized by a relative absence of nuclear activity.

Sosman has pointed out that the cellular arrangements appear to follow an interesting cycle. He observed that the more mature type of foam or xanthoma cell is found towards the periphery, while the small mononuclear and evidently younger class of cell occupies the centre of the lesion—the area, in other words, in which the vessel, the real centre of the disturbance, originally lay. One is left with the impression that the process is one of flow and ebb, the immature cell passing inwards to receive its cholesterol content, and, having done so, passing to the periphery laden with its intracellular burden.

These are the cellular arrangements which constitute the characteristic granulomatous tissue deposit, but as the lesion develops there is a natural tendency towards fibrosis. The cells which previously acted as macrophages become converted into fibroblasts, with the result that the tissue acquires a curious nodular character, an indiscriminate arrangement of fibrous tissue, giant cells, fibroblasts, and lipoid deposits which are partly intracellular and partly free.

Such are the phases of cell activity in the lesion as the microscope reveals them, and I think all will agree that the picture is a remarkable demonstration of the effects which follow the ingestion by the cell of what is usually regarded as a chemically inert substance. Bayliss,[25] in his *Principles of General Physiology*, suggests that the ubiquitous distribution of cholesterol indicates that it plays some important part in the mechanism of the cell, but he does not state what that influence may be. As we view it, the appearances suggest that its existence in any quantity results within the body in some degree of irritation—its presence at least calls into action the great phagocytic function of the reticulo-endothelium, and what is regarded as granulation tissue is but the response of phagocytic elements in their attempt to deal with the invasion. One of the most impressive features in the histopathology is the arrest of karyokinesis; there appears to be no nuclear division in the cholesterol-containing cell, and it may be a significant fact that when the lipoid enters the cell it appears to collect in the perinuclear area. Can it be that its effect in bulk is to arrest nuclear division, and that in amounts which are

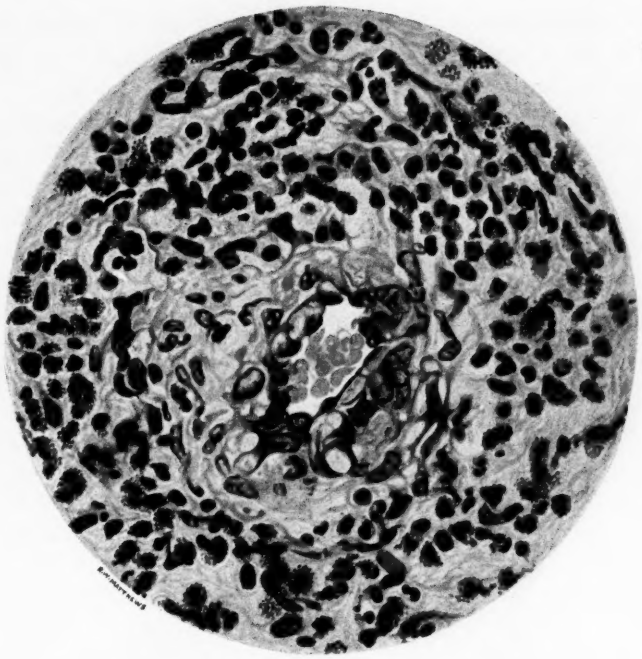

FIG. 567.—Drawing of a section showing the early stage of lipoid granulomatosis. A capillary in cross-section shows the swelling and distortion of the lining endothelial cells. An intracellular accumulation of lipoid is apparent in certain cells. Note the accumulation of lymphocytes and eosinophils in the perivascular area. (× 445.)

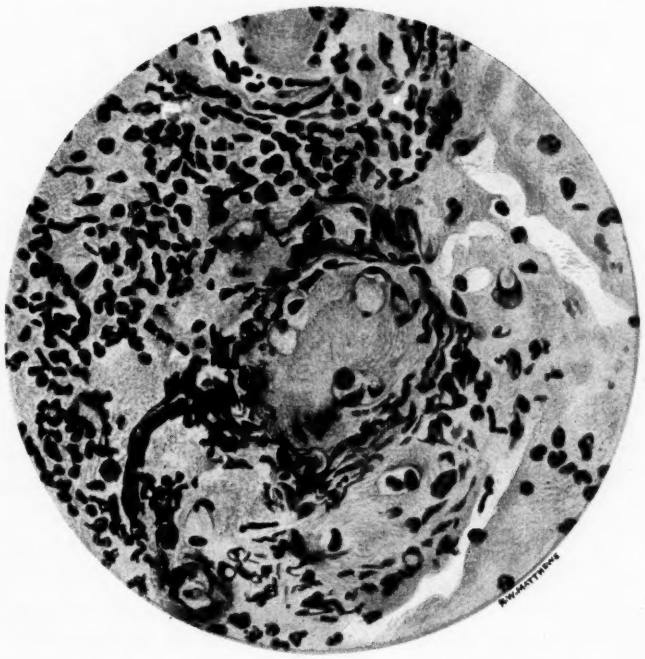

FIG. 568.—Drawing of a section showing the stage of lipoid deposit. The original outline of the capillary is shown. In the centre of the field there is a deposit of lipoid, while a number of lipoid-laden cells which have not yet discharged their contents are seen around the periphery. (× 280.)

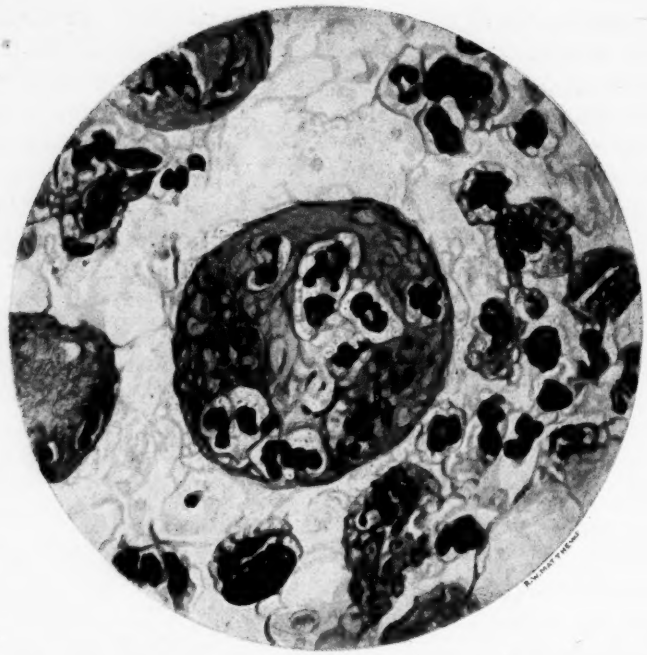

FIG. 569.—Drawing of a section showing the structure of the so-called multinuclear giant cell. The appearance is produced by the fusion of a number of individual lipoid-laden cells. Note the fashion in which the lipoid appears to collect in the perinuclear area. The majority of the surrounding cells are lipoid-laden histiocytes and reticulum cells. (× 630.)

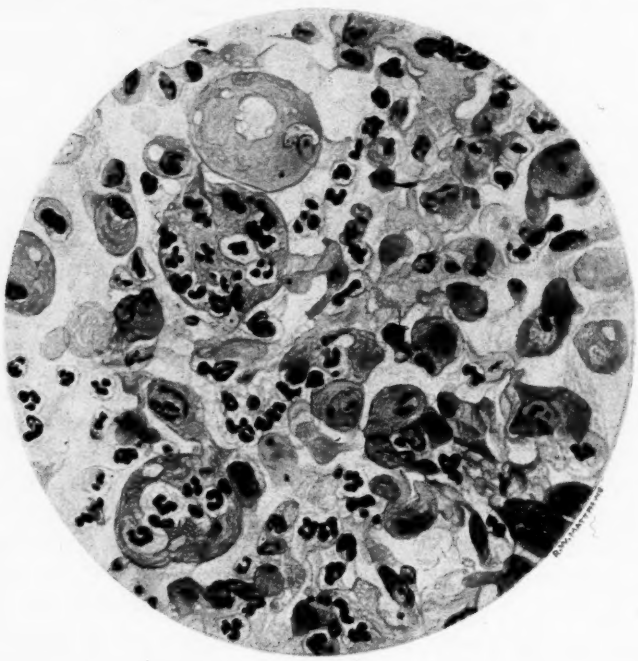

FIG. 570.—Drawing of a section giving an impression of the characteristic cytology of lipoid granulomatosis. There are multinuclear giant cells (syncytial masses) showing lipoid deposit in the perinuclear areas, lipoid-laden histiocytes, and reticulum cells. There are also collections of structureless lipoid. (× 255.)

in keeping with health, it has the effect of controlling cell division? One appreciates that, if this theory could be substantiated, lipoidosis might conceivably have an important bearing upon the influence of the growing tissues, even neoplasmic, where mitosis is abundant.

The Effect of the Granulation Tissue Formation.—The formation of the granulation element is associated with certain untoward results. Its ability to infiltrate is insignificant, but it spreads with relative ease in parts occupied by areolar tissue, in such fat-containing areas as the orbit, and in the perivascular spaces. What appears to be an infiltration of an organ is an extension along perivascular channels, and it is evident that fibrous tissue presents an impenetrable barrier to its invasion. Its progress in bone is marked by a progressive and extensive decalcification of trabecular and later of compact bone, with the result that large portions of the calvaria, clavicles, or mandible (all more or less membrane bones) may

FIG. 571.—The skull base, illustrating the distribution of the granulation tissue deposit in the pituitary fossa, extending along the optic nerves to the orbits, and eventually to the lamina cribrosa of the ethmoid bone.

disappear, and from what we find it seems that the process of bone removal is one of osteolysis or phagocytosis initiated and accomplished by the larger type of multinuclear giant cell, which is universally found at the periphery of the lesions. It is significant that, though large areas of bone may be involved, and the skull vault reduced to a condition which has been described as gelatinous, the underlying dura and the overlying scalp often remain unaffected. Deposits in the basal subdural area are rare except in the space which lies around the hypophysis (*Fig.* 571). This area is so frequently affected that it may be regarded as a site of election, and in some instances the sella turcica is filled with a putty-like mass, with the result that there is displacement of the hypophysis and traction or pressure on its stalk. In a few instances infiltration of the hypophysis and of the hypothalamic area has been described, but such is rare. It is a remarkable feature that, though there may be pronounced accumulation in the sella turcica, there is no invasion of the

basi-sphenoid or of the clinoid processes. From the hypophysial fossa the granulation tissue extends forwards around the carotid vessels, and thence in the tissue around the optic nerve, until it finds itself in the orbit, where factors of space and of fat content permit such accumulation of the deposit that the eyeball is displaced.

The effect of the disturbance in regions other than bone is a displacement process; surrounding elements are pushed aside, compressed, and atrophied, tissue spaces are filled and distorted, but, so far as can be observed, it is only in relation to bone that one witnesses the process of phagocytosis which so intimately affects the skeletal outline.

I need not elaborate the pathology further, for the process is a reproduction in different tissues of the group of changes I have outlined, and, but for variation in situation, there is no difference or distinction between the lesions wherever they may occur.

The summary of the pathology is briefly that in certain tissue areas there is a formation of granulation tissue of distinctive cytology, the result of reactive changes in reticulo-endothelial tissue, the process being a reaction to a cholesterol excess in the blood and body fluids.

THE CLINICAL FEATURES

Various observers have described the disease as being distinguished by a trilogy of clinical features—diabetes insipidus, exophthalmos, and calvarial osseous defects. It is true that these are the most obvious and constant evidences of the disease, but there are variations in the clinical picture, and it is probably more accurate to discuss the clinical evidences on a pathological basis.

Children who are affected by the disease are invariably poorly developed and underweight. The obvious explanation is that metabolism is deranged and the processes of tissue nutrition are upset, but to stop there is to leave the real problem unanswered. There must be some original factor underlying the metabolic disturbance. It has been suggested that as a result of displacement of, and pressure upon, the pituitary, growth functions are inhibited and delayed, or that the hypercholesterolæmia in some way affects nuclear division so that the whole process of tissue growth is retarded. The second evidence of the disease concerns the mental attitude of the child. He is cross, irritable, and most difficult to examine. He cries a great deal, and his mental balance appears to be easily upset. I believe that these changes have an evident explanation. Early in the disease there occurs an epidural formation of granulation tissue, and it seems likely that as a result of such a deposit there is an increase of intracranial tension causing discomfort, pain, restlessness, and irritability. In the intervals of rest the child appears apathetic, with an open mouth and a somewhat listless expression. A further evidence of increasing intracranial tension is seen in the condition of the scalp viens. They are unduly prominent, because the epidural infiltration tends to interfere with the natural evacuation of intracranial venous blood by the basal foramina and orbital fissures, and, in order to compensate for this, the scalp veins are engorged and large.

As soon as the peri-hypophysial infiltration becomes established (and it is at a relatively early stage in the progress of the disease) some of the most characteristic features make their appearance. It will be recalled that the arrangement of the

dura mater in the sella turcica consists of two layers, a periosteal and a visceral, the former adhering to the bone, the latter being raised to form the diaphragma sellæ like a tent over the clinoid processes. Between these layers lies a space occupied by the cavernous sinuses and the pituitary body, these structures being separated from each other by endothelial membranes. It is within this endothelial area that the granulation deposit occurs, and as it increases, it is evident that pressure, tension, and traction influences may be exerted not only upon the pituitary but upon " the pituitary complex "—the pituitary proper, its stalk, and the adjacent hypothalamic region of the brain. The clinical evidence of these disturbing influences is shown in two manifestations—the polydypsia and polyuria of diabetes insipidus, and an infantilism sometimes combined with the adiposo-genital syndrome. The diabetes insipidus manifestation is the more constant, and in respect of priority of symptoms, the first to appear. It is probably due to irritation of the hypothalamic region, the result of tension upon the stalk of the pituitary. Similar symptoms have been produced by experiment and in association with pathological evidence (Greuing[26]), and it is more reasonable to accept this explanation than to assume that they proceed from influence exerted upon and restricted to the posterior lobe of the pituitary. The infantilism and genital syndrome are a later development, and there seems no reason to doubt that these changes arise from disturbances of the anterior lobe, the structure of which is such that perivascular invasion by the granulation tissue may occur.

The explanation of the exophthalmos is apparent. From the region of the pituitary fossa the granulation tissue extends forwards along the endothelial membrane which separates the cavernous sinuses from the pituitary body; it surrounds the optic chiasma, and, passing through the optic foramen and superior orbital fissure, it reaches the orbit, where it accumulates to such an extent that exophthalmos may result. The degree of the disturbance varies—in some cases it is scarcely perceptible, in others (as in a case recorded by Wheeler) the protrusion is so pronounced that it may become necessary to close the palpebral fissure artificially in order to protect the globe.

The calvarial osseous defects have been described in relation to the pathology, and I need not elaborate this point further beyond repeating that the granulation tissue begins in the epidural or intradural area whence it extends into the diploë, probably along the line of entering vessels, for the most constant sites of the original deposits in the bones are at those points in the parietal and occipital bones at which such vessels are encountered. In resistant or untreated cases the calvarial destruction is so complete that the condition fully justifies the description which the French have used—the term ' gelatinous skull '. As I have already indicated, deposits may occur in the clavicles, and they are encountered, though less frequently, within the long bones.

While diabetes insipidus, exophthalmos, and cranial deficiencies may be said to constitute the classical trilogy of clinical features, other evidences may arise according to the area in which the granuloma occurs, and theoretically the changes may appear wherever reticulo-endothelial tissue is encountered. There may be lymph-node enlargement from deposits in lymph-vascular tissue, jaundice may arise from changes relating to the distribution of Kupffer's cells in the liver, there may be signs suggestive of pleurisy or even of pulmonary consolidation, the result of changes in the pleura or in the cells of the alveoli. It comes to this, that the

clinical features are direct manifestations of the pathology, and, in view of the widespread distribution of the tissues which provide the changes, the possible variation in signs and symptoms is considerable.

DIAGNOSIS

As a rule the diagnosis does not present particular difficulty. The disease is no doubt comparatively uncommon, but cases probably occur more frequently than we realize. They are confused with tuberculosis, with tumours of bone, and with primary pituitary disease. Literature records at least one instance in which a child underwent removal of both eyes because those in charge of the case ascribed the exophthalmos to a sarcoma involving the orbital cavities.

In early cases the diagnosis is based upon the following points—the age and general appearance of the child, the occurrence of defects in the calvaria, accompanied, it may be, by the presence of scalp swellings, the development of polyuria and polydypsia, the result of pituitary involvement. In late cases there is dwarfism, exophthalmos, and the adiposo-genital syndrome. The radiographic appearance of the skull bones is often characteristic, the lacunæ showing clear-cut edges with irregular outlines, so that the skull defects may present a map-like or geographical outline, to which the Germans give the name '*Landkartarige*'. The blood readings show an excess of cholesterol, of total acids, and of lecithin. The characteristic histological picture is a proliferation of the reticulo-endothelial cell groups illustrating the presence of large numbers of phagocytic cells, multinucleated giant cells, and the large lipoid-containing or foam cells.

The cytology of the area is characteristic, but here too there are possibilities of confusion, for the picture may be mistaken for that presented by the simple giant-cell tumour, by a myeloma, by tuberculosis, and by syphilis.

PROGNOSIS

The prognosis in large measure is dependent upon the stage at which the disease comes under treatment, but in general it may be said that, while there are no doubt variations in the degree of acuteness of the disturbance, the general tendency is towards an increasing distribution and extent of the disease. A patient referred to by Christian[27] was alive and relatively well at the end of ten years, Schüller's[28] patient was well at the end of sixteen years, one of Sosman's[29] patients enjoyed relatively good health for seventeen years. Rowland[30] collected the results of 14 cases in 1928, of which 7 individuals had died of the disease, while 7 were alive. Sosman[31] reported 9 additional cases in 1931, with 7 survivals. It is our own experience that, if the condition is recognized early, and if appropriate treatment is instituted, the prognosis is relatively good. Of the 3 children who have come under our observation, 2 are alive and well, one at the end of five years, while the third succumbed to an intercurrent infection. It is possible that even in the absence of treatment a tolerance is acquired, or the lesions heal and disappear.

Those who pass to a fatal issue because they have been unrecognized, or because they have failed to respond to treatment, succumb for various reasons—it may be from asthenia and general weakness, or heart failure, or the development of an intercurrent disease reacting on a system which is already debilitated to a serious extent.

TREATMENT

In regard to the question of treatment, certain accepted lines are generally followed. A diet is prescribed which is as far as possible cholesterol-free, but which contains a large percentage of green vegetables. Endocrine therapy in the shape of extract of pituitary is employed, and there is no doubt that this substance controls the diabetes insipidus. Small amounts of thyroid extract were used by Rowland, but apparently without benefit. Irradiated foods, parathyroid gland extract, heliotherapy, cod-liver oil, and calcium have all been tried without success. Sosman has used insulin in the treatment of these cases, giving a dosage of 10 units daily, and he finds that, while it causes improvement in the general health, it apparently has no effect upon the hypercholesterolæmia, nor does it directly influence the granuloma.

The sheet anchor of treatment is deep radiotherapy, and there is no doubt that since this procedure has been followed the results, both immediate and late, have been vastly improved. In our own cases the procedure has been to irradiate the various areas with a dosage of 150 kilowatts 4 milliamperes, each area being irradiated for a period of ten minutes on every third day, a filter of 3 mm. of aluminium being used. The total number of applications varies according to the response—in the last case a series of forty has already been given. The effect of the deep radiotherapy is to destroy the distended and lipoid-laden histiocytes. The lipoid being thus liberated, is disposed of elsewhere, while the original lesion undergoes fibrosis, recalcification, and healing.

SUMMARY

In this paper I have attempted to outline the pathology and the clinical aspects of a significant and interesting disease. There are indications that its incidence is more common than we realize, and the likelihood is that the condition is either overlooked or is misdiagnosed. Perhaps its most outstanding feature is the production of a granulomatous tissue which bears certain resemblances to a malignant tumour, but which arises as the reaction of reticulo-endothelial tissues to the stimulus of a lipoid which is excessive in amount, and which possibly possesses abnormal characteristics. The entire clinical picture is dependent upon disturbance of body tissue and function as the result of the pressure and displacement influences of the granulation deposit.

While the prognosis is always serious, there is evidence that by the use of deep radiotherapy the disease can be checked and even permanently arrested.

In the preparation of this paper I have had a great deal of assistance from Mr. David M. Greig, F.R.C.S.E., LL.D., Curator of the Museum of the Royal College of Surgeons, Edinburgh. It was he who first drew my attention to the significance of the condition, and he has given me great assistance with the literature, and with many problems of the pathology. I am grateful to Professor Woodburn Morison and to Dr. Leila Hawksley for permission to quote a case which has been under their care; they have also allowed me to make use of certain radiograms and photomicrographs illustrative of the case. Mr. John Bruce, F.R.C.S.E., has assisted me in the general preparation of the paper. I am indebted to

Dr. Duncan Whyte, Dr. Allan, and Dr. King for the radiological examination of the cases, and to Mr. Smith for the photographic reproductions.

REFERENCES

1 HAND, A., *Arch. of Pediat.*, 1893, x, 673.
2 SMITH, T., *Trans. Pathol. Soc. Lond.*, 1865, xvi, 224.
3 KAY, T. W., *Pennsyl. Med. Jour.*, 1905–6, ix, 520.
4 SCHÜLLER, A., *Forts. a.d. Geb. d. Röntgenstrahlen*, 1915–6, xxiii, 12.
5 CHRISTIAN, H. A., *Contrib. Med. and Biol. Research*, 1919, i, 390.
6 CHRISTIAN, H. A., *Med. Clin. N. Amer.*, 1920, iii, 847.
7 HAND, A., *Amer. Jour. Med. Sci.*, 1921, clxii, 509.
8 THOMPSON, C. O., KEEGAN, J. J., and DUNN, A. D., *Arch. of Internal. Med.*, 1925, xxxvi, 650.
9 GROSH, L. C., and STIFEL, J. L., *Ibid.*, 1923, xxxi, 76.
10 DENZER, B. S., *Amer. Jour. Dis. Child.*, 1926, xxxi, 480.
11 SCHÜLLER, A., *Brit. Jour. Radiol.*, 1926, xxxi, 156.
12 KYRKLUND, R., *Zeits. f. Kinderheilk.*, 1926, xli, 56.
13 STOWE, W. R., *Trans. Austral. Med. Cong.*, 1927, Sept., 144.
14 ROWLAND, R. S., *Arch. of Internal Med.*, 1928, xlii, 611.
15 HAUSMAN, L., and BROMBERG, W., *Arch. Neurol. and Psychiat.*, 1929, xxi, 1402.
16 CAMERON, A. T., *Text-Book of Biochemistry*, 3rd ed.
17 WILE, U. J., ECKSTEIN, H. C., and CURTIS, A. C., *Arch. Dermatol. and Syph.*, 1929, xix, 35.
18 RAAB, W., *Zeits. f. d. ges. exper. Med.*, 1928, lxii, 366.
19 ANITSCHKOW, N., and CHALATOW, S., *Centralb. f. allg. Pathol. u. pathol. Anat.*, 1913, xxiv, 1.
20 YUASA, D., *Beitr. z. pathol. Anat. u. z. allg. Pathol.*, 1928, lxxx, 570.
21 FONTANA, P., MIMILLA, A., and COLLAGO, J. A., *Ann. de Fac. de Méd. Montevideo*, 1929, xiv, 1075.
22 CHALATOW, S., *Virchow's Arch.*, 1929, cclxii, 691.
23 DAVIDSON, J., TURNER, A. L., and WHITE, A. C., *Edin. Med. Jour.*, 1925, xxxii, 153.
24 SOSMAN, M., *Jour. Amer. Med. Assoc.*, 1932, xcviii, 110.
25 BAYLISS, W. M., *Principles of General Physiology*, 4th ed., 134.
26 GREUING, R., *Die Lebensnerven*, 1924, 78 (edited by R. Müller, Berlin).
27 CHRISTIAN, H. A., *Med. Clin. N. Amer.*, 1920, iii, 847.
28 SCHÜLLER, A., and CHIARI, H., *Wien. klin. Woch.*, 1930, xliii, 153.
29 SOSMAN, M., Loc. sup. cit.
30 ROWLAND, R. S., *Arch. of Internal Med.*, 1928, xlii, 611.
31 SOSMAN, M., *Amer. Jour. Roentgenol.*, 1930, xxiii, 581.

AN UNUSUAL CASE OF BONE REGENERATION AFTER COMPLETE DIAPHYSECTOMY ON TWO OCCASIONS*

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REGENERATION of the diaphysis of a bone such as the tibia on two occasions after the shafts (the original and the new) had been removed on account of acute disease is unusual and worthy of record. More particularly is this so when the new shafts formed resembled the normal bone in size and shape.

The patient—a boy of 6 years—gave a history of having fallen heavily on the right leg three days before coming into hospital. The day before admission he was fevered, and had severe pain and tenderness in the leg. On examination the right leg was swollen and œdematous; there was much tenderness at the junction of the middle and lower thirds of the tibia, and the child appeared acutely ill. At operation a diffuse osteomyelitis and extensive necrosis were found in the tibial diaphysis, which was removed completely. The opening in the periosteum through which the bone was removed was closed with sutures. The shaft removed subperiosteally was completely necrotic, and scattered irregularly over the surface were patches of congestion (*Fig.* 572). Where the shaft had been in apposition with the epiphysial plates at the upper and lower ends there was some irregular absorption of bone; at the lower end this absorption formed a dome-shaped depression. Radiological examination four days afterwards showed complete absence of the tibial diaphysis (*Fig.* 573); six weeks later there was extensive formation of new bone (*Fig.* 574). The child was able to walk six months after operation, and radiologically the bone, though somewhat irregular in outline, appeared to have completely re-formed (*Fig.* 575).

The boy remained in good health for two and a half years after dismissal, when he was readmitted complaining of pain over the scar below the right tibial tuberosity. There was a history of slight trauma in this region some days previously. On an incision being made through the old scar, a large quantity of sanguineous pus was evacuated from under the periosteum, and carious bone was removed from the medullary cavity. Four days later there was much swelling in the region of the knee, and the child was gravely ill. An incision was made down the front of the tibia from knee to ankle, and on opening the periosteum an extensive osteitis of the whole shaft was found. To facilitate removal of the bone, the shaft was cut through below the middle, and both upper and lower parts of the diaphysis were excised.

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FIG. 572.—Posterior surface of original shaft; irregular absorption of bone at upper and lower ends. ($\times \frac{5}{16}$.)

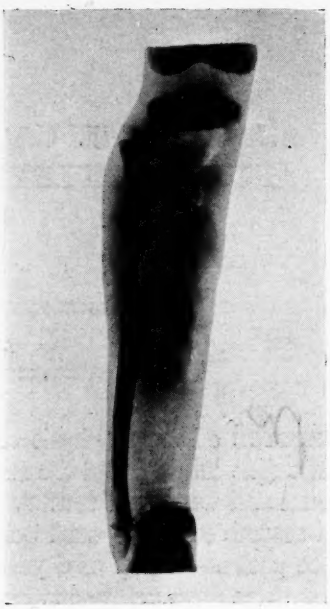

FIG. 573.—X-ray plate four days after complete removal of tibial diaphysis.

FIG. 574.—X-ray plate six weeks after operation; partial regeneration of tibial diaphysis.

FIG. 575.—X-ray plate six months after operation; complete regeneration of tibial diaphysis.

This second shaft had developed from the periosteum stripped from the original shaft removed three years previously, and though extensively necrotic, was somewhat remarkable (*Figs.* 576, 577). It was 4·5 cm. longer than the original and also thicker, while the various markings were more prominent, and in spite of some irregularity the new bone showed the general anatomical features of the normal bone for a child of this age, even to the position of the foramen for the nutrient artery at the junction of the upper and middle thirds of the posterior surface. Thus growth of the bone corresponding to the general growth of the child had taken place.

A large area of necrotic bone had been removed surgically at the upper end of the medial aspect, where the line of separation from the epiphysial plate was very

FIG. 576.

FIG. 577.

FIG. 578.

FIG. 576.—Posterior surface of second shaft. The bone is thicker than normal and more irregular in outline. The markings for the muscular insertions are fairly well defined and the foramen for the nutrient artery is present at the junction of the upper and middle thirds. The bone was divided transversely near the lower end to allow of easier removal. ($\times \frac{12}{41}$.)

FIG. 577.—Medial aspect of second shaft showing area of necrosis below medial condyle. ($\times \frac{12}{41}$.)

FIG. 578.—Anterior aspect of third shaft after amputation. The large abscess cavity extending into the head of the tibia is seen. ($\times \frac{13}{45}$.)

irregular (*Fig.* 577). A perforation was present in the upper epiphysial plate, and through this infection had reached the knee-joint. The whole wound was thoroughly dressed, and the knee-joint washed out with eusol and a drain inserted. Repeated dressings were necessary during the month following the operation, at the end of which radiological examination showed new bone formation. During the next month the condition of the knee-joint did not improve, the temperature remained high, and the child's condition became more serious. Consequently, it was considered advisable to amputate the leg. Dissection of the specimen after amputation showed an irregular perforation in the head of the bone leading to the knee-joint, in which there was an extensive purulent arthritis, with destruction of cartilage and

synovial membrane. The tibial diaphysis had partly regrown (*Fig.* 578) and consisted for the most part of osteoid tissue, which showed slight superficial sepsis. This third shaft had formed in ten weeks after removal of the second. After the amputation the child's condition quickly improved, and he was able to go about on crutches at the end of six weeks.

DISCUSSION

Opinion is divided as to the advisability of complete removal of a necrotic diaphysis in cases of acute osteomyelitis. In the case reported complete restoration of the part justified the removal of the whole of the dead shaft. This, however, may not always occur, and probably depends on the acuteness of the infection. The drawing together of the periosteum near the upper epiphysial line was probably a technical error, as it allowed an unavoidable pocket to remain, in which latent infection persisted owing to lack of efficient drainage and gave trouble at a later date. Owing to the great vegetative capacity of the osteogenic tissue in the early years of life the diaphysis in this case was on two occasions restored, in the first instance completely, and in the second only partially. Further, the new bone in the first replacement corresponded closely in its structural characters, size, and shape to that in the normal child. This was probably due to the limiting action of the periosteum, together with the moulding effect of the surrounding muscles.

SUMMARY

A short account is given of a case of osteomyelitis of the tibia, with regeneration of the shaft after removal of the original bone and also after excision of the new shaft in which osteomyelitis recurred.

THE TREATMENT OF SPASMODIC DYSPHAGIA BY SYMPATHETIC DENERVATION*

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Dr. D. R. Paterson, of Cardiff, was one of the first, if not the first, to describe a now well-known syndrome occurring in women and comprising spasmodic dysphagia, anæmia, and atrophic changes in certain mucous membranes, and to point out the highly significant fact that there is a tendency for patients with this condition ultimately to develop pharyngeal carcinoma. In 1919 Paterson reported his observations, which had been made during the course of many years, to the Laryngological Section of the Royal Society of Medicine.[1]

Hitherto the treatment of this condition of spasmodic dysphagia has been largely symptomatic,—exhibition of iron for the anæmia, dilatation of the resistant sphincter at the lower end of the pharynx, and surgical or radium treatment for carcinoma when this supervened.

In discussing the syndrome with my colleague, Mr. R. D. Owen,[2] who each year sees a number of these cases in his Laryngological Department, it occurred to me that the condition might be alleviated and the possible onset of carcinoma prevented if two desiderata could be established—namely, relaxation of the sphincter (supraœsophageal sphincter of Sir William Turner, horizontal part of inferior constrictor, crico-pharyngeus), and secondly, an increase in the blood-supply to the hypopharyngeal mucosa, examination having repeatedly shown that an anæmic and atrophic condition of this mucosa existed in association with tight constriction of the sphincter. Observation had further shown that as these cases progressed the mucosa became damaged by the passage of food and instruments, and changed in character† until ultimately metaplasia resulted in the terminal event, hypopharyngeal carcinoma.

It appeared that we might be able to relax the sphincter and improve the blood-supply if we could remove the sympathetic innervation of the sphincter and the lower part of the pharynx, and investigation showed that the sympathetic component of the pharyngeal plexus was derived entirely from the superior cervical ganglion. It seemed, therefore, logical to perform bilateral superior cervical ganglionectomy to bring about our object (*Fig.* 579).

At this time Dr. Ivor Davies had one of these cases under his care in a medical ward, and he readily concurred with the suggestion of Mr. R. D. Owen and myself that if the patient consented she should be given the chance of relief which the contemplated procedure appeared to offer. Accordingly, her consent having been

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† Paterson described this change as "a thinning of the superficial epidermal layer and an apparent thickening, from infiltration, of the underlying tunica propria".

obtained, the left superior cervical ganglion was removed, and eleven days later the right one also. *Figs.* 580–584 show the ganglia and their microscopical appearances, and the patient with a double Horner's syndrome (enophthalmos and myosis)

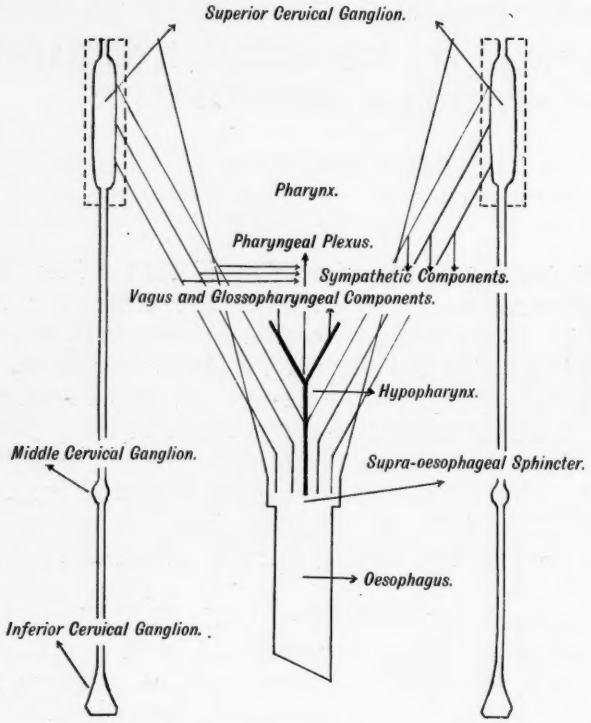

FIG. 579. — Diagrammatic representation of the sympathetic innervation of the pharyngeal plexus, showing how bilateral superior cervical ganglionectomy destroys the sympathetic innervation.

FIG. 580.—Right superior cervical ganglion.

FIG. 581.—Left superior cervical ganglion.

consequent upon destroying the sympathetic fibres to the orbit. The Horner's syndrome does not inconvenience her in any way, in fact, vision is better through the small pupil; nor, since it is bilateral, does it detract from her appearance. She

is able to swallow food of any kind, and expresses herself very satisfied with the operation.

A brief account of the case is as follows :—

A married woman, aged 59, the mother of three healthy children, complained of difficulty in swallowing of two and a half years' duration and of attacks of slight breathlessness. In May, 1932, she first had difficulty in swallowing solid food, but had noticed that if she drank large quantities of water with her food, she could to a certain extent overcome the difficulty. The food appeared to stick at the suprasternal notch or rather above it, and there was an occasional burning sensation when attempting to swallow.

FIG. 582.—Histological appearance of right superior cervical ganglion.

FIG. 583.—Histological appearance of left superior cervical ganglion.

On Sept. 5 she was examined by direct œsophagoscopy by Mr. R. D. Owen, who found post-cricoid constriction and had difficulty in passing the tube behind the cricroid—the mucosa was cracked. Following instrumental dilatation she had some relief.

On Dec. 30 œsophagoscopy was again performed, and the tube passed easily. The condition then relapsed, and on Oct. 14, 1934, she was admitted to the Cardiff Royal Infirmary under the care of Dr. Ivor Davies. Œsophagoscopy now showed slight post-cricoid hyperplasia but no evidence of new growth, the mucosa was more resilient than on previous occasions, there was less anæmia, and no cracking took place when the œsophagus was entered. A test-meal on March 1 showed achlorhydria.

On Sept 7, 1932, her erythrocytes were 3,760,000 and the hæmoglobin 65 per cent; on Sept. 27, the figures were 3,040,000 and 50 per cent; on March 27, 1934,

4,240,000 and 45 per cent; on Oct. 10, 4,529,000 and 54 per cent; on Oct. 16, 5,808, 000 and 64 per cent; and on Nov. 3, 5,420,000 and 65 per cent.

Examination showed a rather spare woman with a sallow complexion and a smooth tongue.

OPERATION.—On Nov. 22, 1934, the left superior cervical ganglion was removed through a curved incision extending from behind the mastoid process downwards and forwards across the anterior part of the sternomastoid, which muscle was retracted laterally and the carotid sheath exposed. This was opened and the vagus recognized and retracted. The ganglion was identified lying behind the sheath on the rectus capitis anticus major and longus colli muscles. The superior cardiac nerve was noted as the largest branch of the ganglion and was divided. The branches to the pharyngeal plexus were divided and the ganglion removed (*see Fig.* 581), with its connecting sympathetic cord below its lower pole. The deep fascia and platysma were closed with catgut, and the skin with clips.

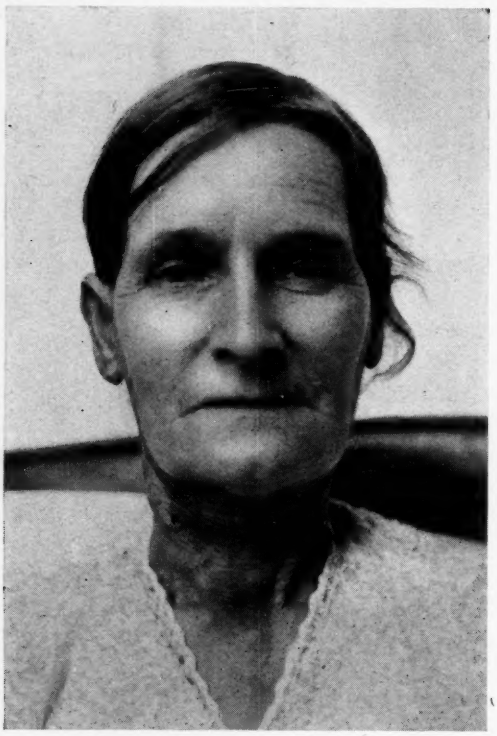

FIG. 584.—Patient ten days after second operation. Bilateral Horner's syndrome apparent.

A similar operation was performed on Dec. 3, 1934, on the right side, and the ganglion removed. (*See Fig.* 580.)

Fig. 584 shows the condition of the patient with bilateral Horner's syndrome ten days after the second operation was performed.

I have sought early publication because these cases, though not very common, are from time to time met with in most laryngological or medical out-patient departments, and as the procedure described appears to be a logical way of dealing with them, it would seem desirable to give these unfortunate sufferers such chance as it offers. Apart from immediate relief it may obviate the onset of carcinoma. It remains for time to show whether relapses will occur, and I shall welcome the reports of others who may accept the line of reasoning submitted and be prepared to give this procedure fair trial.

BIBLIOGRAPHY

[1] PATERSON, D. R., *Jour. of Laryngol.*, 1919, xxxiv, 289.
[2] OWEN, R. D., Personal communication, 1934.

ON OSTEO-ARTHRITIS IN THE DORSAL INTERVERTEBRAL JOINTS

A STUDY IN MORBID ANATOMY

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INTRODUCTION

The material on which the following observations have been made consists of nothing but dried macerated bones derived from vertebral columns assembled for anatomical or anthropological research. It is greatly to be regretted that no clinical notes of any sort are available.

The purpose of this paper is to describe the occurrence of osteo-arthritis in the synovial joints of the human vertebral column, and to offer speculations on the circumstances which cause certain regions of the vertebral column to be particularly prone to the development of this disease and others to be less so.

From the outset it will be well to state plainly that the condition commonly called 'osteo-arthritis of the spine' is not the subject of discussion in this paper. The term 'osteo-arthritis of the spine' is, I think, never used to convey a meaning which is comparable with that conveyed by 'osteo-arthritis' of, say, the knee or shoulder. In osteo-arthritis of the great movable weight-bearing joints of the body no single character is so constant as the presence of osteophytes, which, at least in their early stages, grow from the edges of the areas of contact and weight bearing. In the vertebral column of man no bony disease is more common than that marked by the growth of osteophytes from the edges of adjacent vertebral bodies towards each other. By analogy, as I assume, and in the absence of precise information as to morbid anatomy, growth of osteophytes has seemed to justify the use of the name 'osteo-arthritis of the spine' for this disease.

The joint between a pair of vertebral bodies is formed by layers of fibrous tissue built upon the flat cartilage-covered surfaces of the vertebral bodies, and containing neither cavity nor synovial membrane, but a mass of semi-fluid mucin-like matter under pressure. The synovial joints of the body generally present a contrast in that each has all the following features—a cavity, cartilage-covered contact surfaces, and a vascular synovial membrane, all enclosed in a fibrous capsule.

It is in movable, synovial, or diarthroidal, joints that osteo-arthritis has been studied by a host of observers from the clinical, the pathological, and the experimental points of view. There can be no doubt that osteo-arthritis as ordinarily regarded is exclusively a disease of synovial joints.

Each unit of the vertebral column articulates with each of its neighbours by its dorsal arch and by its body. The joints between the vertebral bodies are of the sort briefly described above and ordinarily called the 'intervertebral discs' or 'fibrocartilages'. Each dorsal arch sends upwards a pair of superior articular

processes, and downwards a pair of inferior articular processes. By contact of the superior articular processes with the inferior articular processes of the arch of the vertebra placed next headward, and vice versa, is built up a chain of joints between the dorsal arches of the vertebræ. These are the dorsal intervertebral joints, and are synovial in character. It is osteo-arthritis of these dorsal intervertebral joints which is the subject matter of this paper.

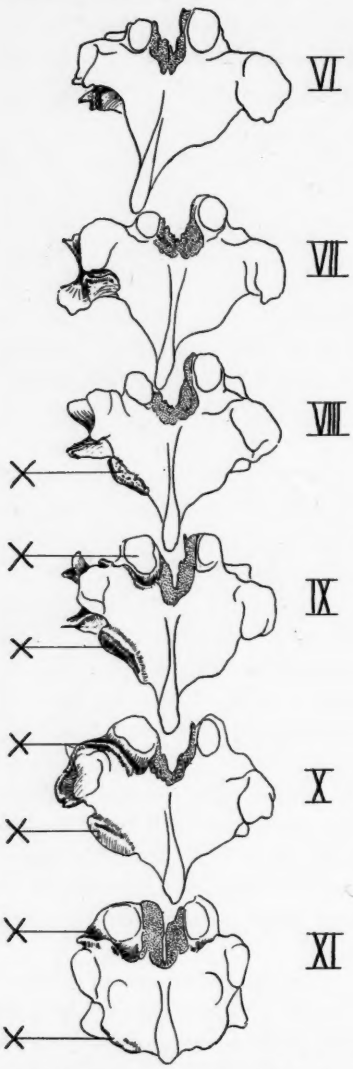

FIG. 585.—A posterior view of the dorsal arches of six thoracic vertebræ set out in descending series. X indicates the presence of osteo-arthritis in the dorsal intervertebral joint. Note that this change affects the dorsal intervertebral joints on the *left side only*. These vertebræ were found in the hollow of the scoliotic column whose concavity lies towards the left in this region, as is shown in *Fig.* 592.

Distribution in the vertebral column of osteo-arthritis of the dorsal intervertebral joints is not stated in any printed work to which I have access; indeed, in only two places have I met any reference to osteo-arthritis of these joints at all. The joints between the vertebral bodies are far larger than those between the dorsal arches, and are far more readily displayed by X-rays. This may be the reason why disease in the dorsal intervertebral joints is commonly overlooked and such clinical significance as disease in this position may have is not appreciated. Other synovial joints in the vertebral column are those made by the ribs, the costo-central and the costo-transverse, the occipito-atloid, and the anterior atlanto-axoid joints.

Robert Adams, of Dublin, is one of the earliest modern writers on osteo-arthritis. His book, *A Treatise on Chronic Rheumatic Arthritis* is entirely devoted to a study of osteo-arthritis—to use the appropriate modern term—in most of the important synovial joints of the body. The author gives an account of osteo-arthritis in the dorsal intervertebral joints and, curiously, in the anterior atlanto-axoid joint[1]; but he entirely disregards the common osteophytic disease of the vertebral bodies to which the name 'osteo-arthritis of the spine' is so frequently applied. Arbuthnot Lane[2] records disease in these joints and also in the anterior atlanto-axoid joint in his account of the changes in the vertebral column which accompany heavy labour.

THE CAUSES OF OSTEO-ARTHRITIS

That mechanical forces have a large share in the production of this disease will probably be universally admitted, and in the vertebral column this can be readily shown by the details of a common occurrence.

The dorsal intervertebral joints in the cavity or pressure zone of a scoliotic spine are likely to acquire the disease. *Fig.* 585 shows osteo-arthritic changes in a series of joints, all on the left side, derived from the concavity of a scoliotic spine

(the same as is shown in *Fig.* 592). When disease grossly disturbs the normal balance of the vertebral column an almost invariable associated finding is osteo-arthritis in the dorsal joints above and below the district of disturbance.

Fig. 586 shows osteo-arthritis in the dorsal intervertebral joints above and below the region of gross disturbance caused by collapse of a vertebral body. The link of association must be mechanical strain. Among the causes of osteo-arthritis Timbrell Fisher[3] notes: "a localized increase of articular stress of an occupational nature or caused by mal-united fracture, disease of the limb bones with consequent deformity, or ankylosis of an adjacent joint". The principle underlying this generalization is contained in the observations I have made above. In this connection the strongly expressed view of Arbuthnot Lane[4] is worth recalling: "There is no such disease as rheumatoid arthritis, but the conditions which are regarded as constituting that disease are identical with the changes produced in the osseous system by pressure." When it is noted that the 'rheumatoid arthritis' of this passage is

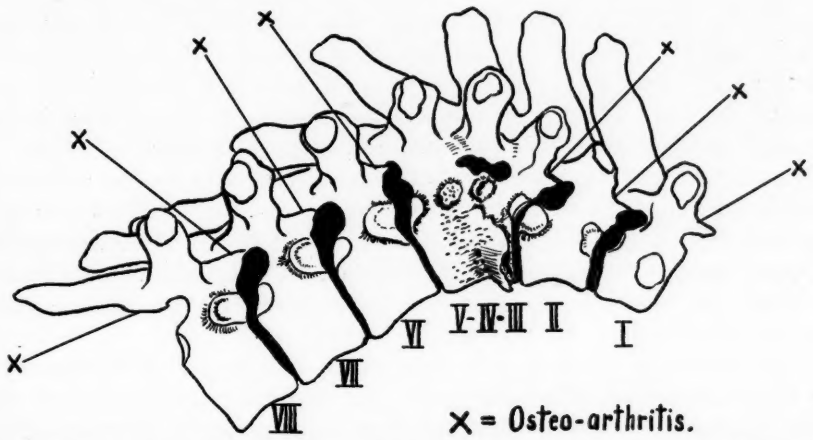

FIG. 586.—Part of a thoracic column viewed from the right side. The 3rd, 4th, and 5th vertebræ have coalesced into one mass following collapse of the 4th vertebra as a result of disease, possibly tuberculous. Angulation of the dorsal column has followed, and osteo-arthritis affects the dorsal intervertebral joints above and below the site of disturbance.

the 'osteo-arthritis' of modern use, a comparison with a recent definition is interesting. Osteo-arthritis is defined by Fisher[5] as: "a series of physiological and pathological changes occurring in a joint subjected to prolonged or oft-repeated injury, either mechanical or toxic, but of a moderate degree of intensity".

Osteo-arthritis might not unfairly be held to represent an effect of wear and tear on a joint. There seems no doubt that central degeneration of articular cartilage is the starting-point of the chain of events that has osteo-arthritis at the other. Under surgical conditions Timbrell Fisher erased the central part of one articular cartilage of a joint in a living animal. The results include degeneration of a corresponding area of the opposing cartilage and the growth of peri-articular osteophytes. It should follow that growth of chondrosteophytes from the two articular surfaces must progress equally, because failure of cartilage contact in any place would lead to degeneration opposite to it; certainly I have found that osteo-arthritis is equally marked in both the bones that enter into a joint.

It might be surmised that each synovial joint has its normal or optimum

provision of cartilage. As cartilage is lost centrally by disease, injury, or wear, the remainder is supplemented by peripheral growth in an attempt to restore the normal allowance. It is growth of cartilage with its scaffolding of bone that constitutes the chondrosteophyte characteristic of the disease, and this takes place at the most vascular region, the periphery.

It is not believed by modern observers that mechanical factors are the only ones that enter into the etiology of osteo-arthritis. Timbrell Fisher, no less than other writers on this topic from the clinical aspect, insists on the need for elimination of any source of toxic absorption in the treatment of a patient. The suggestion is that microbes or toxins may render the cartilages or the bones more open to the influence of pressure.

It is not intended in this place to attempt to give even a brief review of the evidence that the products of microbic activity, disordered metabolism, and internal secretion may each have their influence. It will suffice to remark that osteo-arthritis has been noted in a hæmophilic child aged 13 years,[6] and also in a child aged 15, suffering from premature and imperfect general development—progeria.[7] Garrod[8] has remarked on the " peculiar liability of persons with alcaptonuria and ochronosis to develop osteo-arthritic lesions in later life ".

The mechanical factor is evident and denied by none. For the present purpose this factor is the only one that my material allows me to consider.

I can only presume that pressure between joint surfaces over an extended period is a circumstance likely to result in osteo-arthritis. In the vertebral column, whose function is to bear weight in the erect attitude, this amounts to dorsiflexion between the vertebral units. In dorsiflexion the articular surfaces of the joints borne on the dorsal arches are evidently brought into contact, and in ventriflexion these joint surfaces are relieved of direct pressure.

In the following pages I shall present to the reader observations of facts relating to the occurrence of osteo-arthritis, and shall offer speculations on the means by which mechanical pressure might exert an influence in the production of osteo-arthritis in the dorsal intervertebral joints.

ANATOMY OF OSTEO-ARTHRITIS AS SEEN IN THE DORSAL INTERVERTEBRAL JOINTS

In my survey, so long as peripheral osteophytes could be recognized, the joint in question was regarded as the subject of osteo-arthritis. In advanced states the diseased joint surfaces may be grooved, eburnated, and greatly expanded by the formation of new bone.

In a large number of vertebræ will be found all of the following : bones of which the articular contact surfaces are clearly marked and the evidence of osteo-arthritis is limited to circumferential osteophytes ; bones whose contact surfaces having no vestige of normal structure present irregular surfaces, here polished, there porous, as if treated first with a rasp and then with a burnisher, but still defined by heaped osteophytes around; and every stage between these two extremes.

In *Fig.* 587 is shown a photograph, and in *Fig.* 588 an enlarged scale drawing, of the surface of a flat intervertebral joint moderately advanced in osteo-arthritis. Three areas which are approximately circular and concentric can be recognized :—

Area A.—A central area which corresponds to the cartilage-covered area of a normal joint. This area encloses bone of fine texture and is neither porous nor polished.

Area B.—An intermediate ring of porous bone marked by holes which vary considerably in size and depth. Many of these holes would admit a pin, some are shallow pits, and others penetrate the bone deeply.

Area C.—A third zone consists of a rampart of irregular osteophytes often

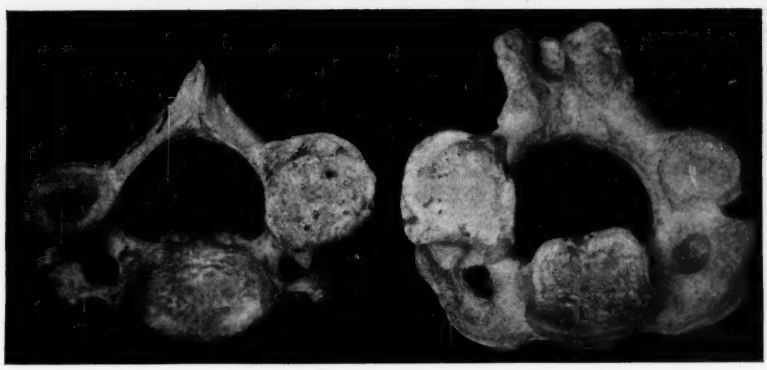

FIG. 587.—The photograph on the right is of the lower aspect of a specimen of the 2nd cervical vertebra. On the left is shown the upper aspect of the 3rd cervical vertebra with which it makes contact. Note the osteo-arthritic joint on the left side of the vertebræ, which is portrayed in more detail in a line enlargement in *Fig.* 588. The spreading osteophytes from the diseased joint, it may be observed, go far to obstruct the groove for exit of the third cervical nerve.

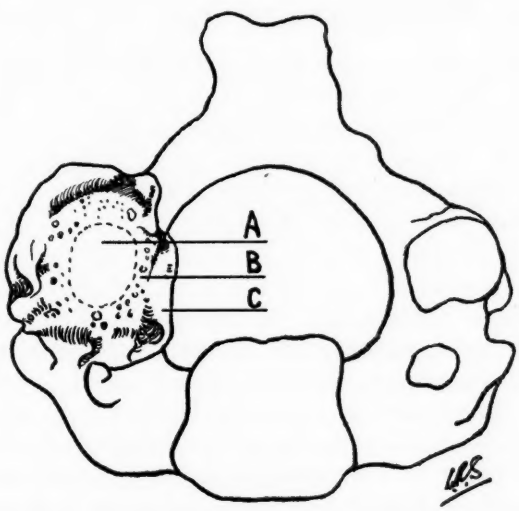

FIG. 588.—Enlarged line drawing of the upper surface of the second left intervertebral joint shown in *Fig.* 587. Note the three areas shown: A, Primary contact area; B, Zone of porous bone; C, Rampart.

discontinuous but encircling the intermediate ring and conspicuous chiefly by their irregularity. These osteophytes are seemingly not of porous bone, but are often highly polished.

By the characters of the contact surfaces it is possible to classify osteo-arthritis

of the small and simple intervertebral joints in three stages, which I conceive to represent consecutive steps in the development of osteo-arthritis :—

Stage I.—The primary contact *Area A* bears osteophytes at its margin. Only *Areas A* and *C* are present, and it must be supposed that expansion of the contact area has only just begun.

Stage II.—All three areas, *A*, *B*, and *C*, are present. Expansion has gone a stage further, and the area of secondary expansion, *B*, is exposed as the rampart recedes. The fact that this *Area B* is porous and sometimes shows blood staining in recently prepared bones, suggests that it is a vascular region. It is probable that the spaces of the vascular bone-marrow are being exposed.

Stage III.—The limits of *Area A* are no longer to be seen. The contact area may be all of porous bone or it may be grooved and eburnated. The rampart *C* may be very conspicuous, or it may, on the contrary, be broken down and worn flush. Eburnation may be found in any part of the articular area at any stage.

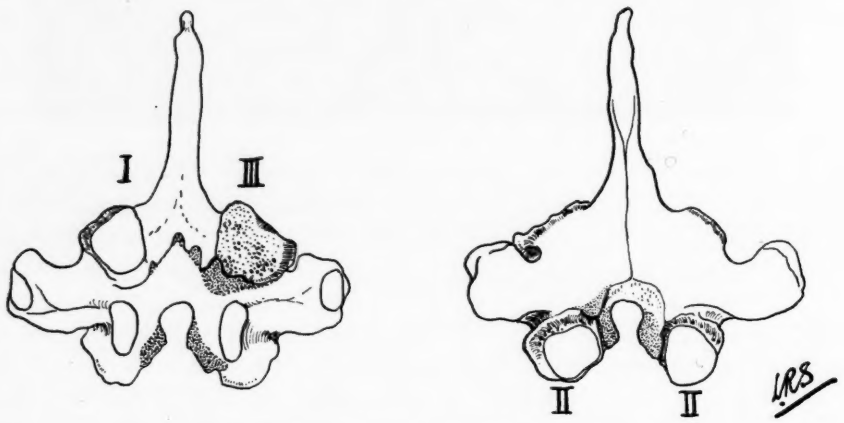

FIG. 589.—Drawings of the dorsal arch of a specimen of the 7th thoracic vertebra. The dorsal arch is viewed from the dorsal aspect in the right-hand drawing and from the ventral aspect in the left-hand. By each of the four articular facets is placed a Roman numeral which indicates the stage of development of osteo-arthritis.

Examples of these three stages are shown in the drawings in *Fig.* 589. Timbrell Fisher, under circumstances which are not available to me, has observed osteo-arthritis in all its aspects, and has based on his pathological findings a classification[9] which I set out for comparison with mine. Fisher's classification, being based on post-mortem observations of very wide range, is far more complete than mine, which is based on a material restricted to dried bones.

The persistence of the normal contact area of a joint is very striking. Though the surface of actual contact is doubled, still this area may be recognizable. An explanation is perhaps to be found in Fisher's [10] observation that "the layer of bone underneath the articular cartilage can be seen to be thickened before the cartilage covering it has entirely disappeared ".

It may be remarked that the small dorsal intervertebral joints are peculiarly suitable for observations of the sort I have made. The articular surfaces are small but even in outline, most of them are flat, and the rest only slightly curved.

Timbrell Fisher's Classification	Classification based on Pathological Anatomy of Bone (*supra*)
Class I: Fibrillation and earlier changes in the central area of the articular cartilage	—
Class II: Fibrillation more marked, commencing lipping of articular margins, with hyperplasia of the synovial membrane	*Stage I*
Class III: Changes more marked, commencing exposure of sub-articular bone, and more generalized changes in synovial membrane. Deformity from chondro-osteophytic formations	*Stage II*
Class IV: Bone extensively exposed, often eburnated and grooved. Destruction of intra-articular ligaments. Synovial fringes usually fibrosed or atrophic. Deformity increased by shortening or subluxation	*Stage III*

THE DISTRIBUTION OF OSTEO-ARTHRITIS OF THE DORSAL JOINTS IN THE VERTEBRAL COLUMN

The material examined includes 126 human vertebral columns, all dried and macerated. These columns, though assembled, have not always been complete in every member. The total numbers for the several vertebræ vary from 90 to 125. In order to present the true proportional incidence of osteo-arthritis on the different parts of the vertebral column, I have made a series of simple calculations and have set out the result in a graph (*Fig.* 590) as if the total number of columns were 100. I examined all the available vertebral columns and noted the joints which displayed clear evidence of osteo-arthritis. In my notes I did not distinguish the stages of development which I have described, but merely recorded the presence of osteo-arthritis. It will be seen from *Fig.* 590 that osteo-arthritis affects the dorsal intervertebral joints in three main 'outcrops', which may be defined and named as follows.

1. Lumbo-dorsal Outcrop.—A fairly regular curve extending from the 8th thoracic vertebra to the lumbo-sacral junction.

2. Dorso-cervical Outcrop.—Irregular in form and including the joints between the 6th cervical and the 7th thoracic units. Two peaks of especially high incidence occur: (*a*) At the joint between the 4th and 5th thoracic vertebræ. This may be called the *upper thoracic peak.* (*b*) At the cervico-thoracic junction, the joint between the 7th cervical and the 1st thoracic vertebræ. This may be called the *cervico-thoracic peak.*

3. Cervical Outcrop.—A curve of fairly regular form between the 1st and the 7th cervical vertebræ.

Commentary on Distribution.—It is necessary to inquire why osteo-arthritis has an unequal incidence on the dorsal joints of different districts of the vertebral column. In general, osteo-arthritis of the dorsal intervertebral joints assumes a higher incidence as the column is followed downwards. This suggests that weight-bearing is an important causative factor, but the presence of maximum and minimum zones shows the need for further examination of the mechanical

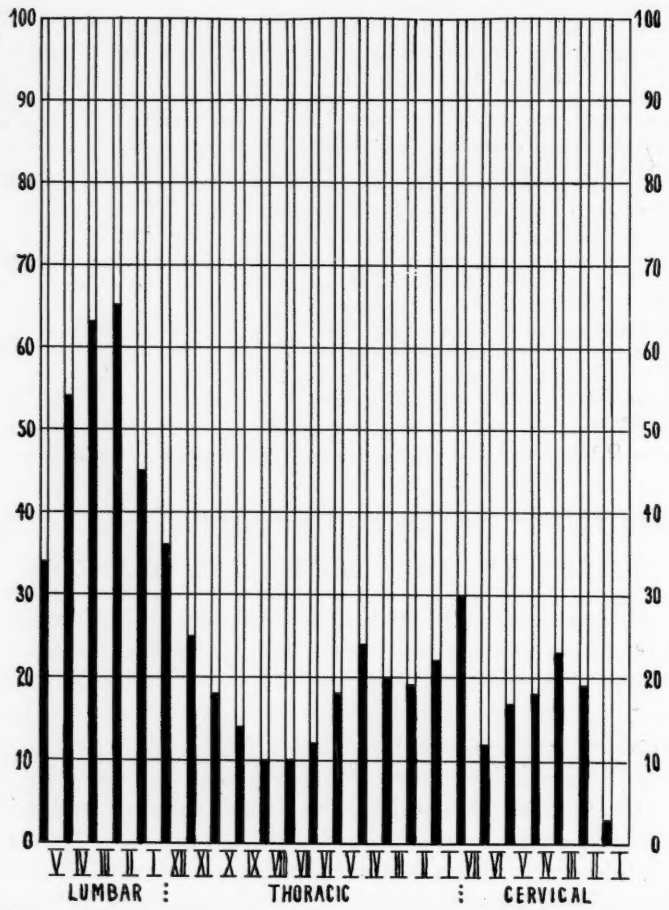

FIG. 590.—Graph showing the distribution of osteo-arthritis of the dorsal intervertebral joints in the vertebral column. The abscissæ represent the vertebræ set out from left to right in ascending order. The ordinates show the proportional incidence of osteo-arthritis on the various joints as if the total number of joints examined were 100. Note the three main ' outcrops '—lumbo-dorsal, dorso-cervical, and cervical—which are separated by zones of minimal incidence at the joints between the 7th and 9th thoracic and at the joint between the 6th and 7th cervical vertebræ. Note the upper thoracic peak at the joint between the 4th and 5th thoracic vertebræ, and the cervico-thoracic peak at the cervico-thoracic junction.

circumstances under which the vertebral column carries weight. The condition under which a given joint bears a direct share in weight-bearing is that of dorsi-flexion, and the presence of dorsiflexion lies in the architecture of the vertebral column. Humphry[11] writes on the alternating antero-posterior curves formed by the mobile units of the vertebral column as follows: " The three upper curves—the cervical, dorsal, and lumbar—are so arranged that their chords are in the same vertical line in the erect position of the body; and that vertical line corresponds with

the line of gravity of the head. . . . The centre of gravity of the cranium with its contents . . . is placed immediately over the heads of the thigh bones; and the points of confluence of three of the intermediate curves are in the line of gravity". It follows from this statement that the units of the cervical and of the lumbar curves are dorsiflexed upon each other, lying as they do in front of the line of gravity and weight-bearing. Hence it follows that the dorsal intervertebral joints in these two curves take a direct share in bearing the weight of the column.

In the thoracic curve, however, the line of gravity lies in front of the vertebræ, which are therefore ventriflexed upon each other. Under weight-bearing the tendency is for the dorsal arches to fly apart. The dorsal intervertebral joints do not ordinarily carry weight in the same way as do those in the cervical and the lumbar curves. On the contrary, in the thoracic curve the dorsal arches are restrained from separation by the interlaminar ligaments—the ligamenta subflava. In another place[12] I have shown that ossification commonly occurs in these interlaminar ligaments and that this occurrence is almost confined to the thoracic region of the spine; I suggest that this effect is the result of strain.

We will consider seriatim the causes of the three outcrops of osteo-arthritis in the dorsal intervertebral joints to which reference has been made.

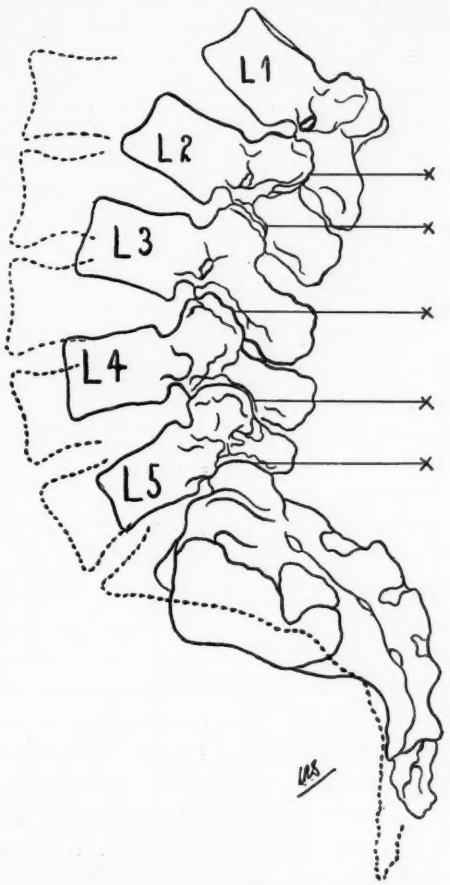

FIG. 591.—Tracing of the lumbar vertebræ with the sacrum reconstructed and viewed in a true lateral position. The dorsal joints indicated by X are all affected with osteo-arthritis. For the purposes of comparison the contour of the corresponding units of a normal column has been superimposed in dotted outline. This superimposed contour was traced from one of Humphry's normal specimens preserved in the Anatomy Museum at Cambridge. It is to be inferred that osteo-arthritis of the dorsal intervertebral joints leads to accentuation of the lumbar curve, i.e., to lordosis.

THE LUMBO-DORSAL OUTCROP OF OSTEO-ARTHRITIS

This outcrop of osteo-arthritis commences as a fairly even curve below the 8th thoracic vertebra, rises to a maximum at the joint between the 2nd and 3rd lumbar vertebræ, and falls away to the lumbo-sacral junction. This outcrop extends over more vertebræ in a headward direction than are ordinarily included in the lumbar curve. In height this curve of incidence exceeds any other, and therefore may be presumed to indicate the site of first development of osteo-arthritis in the dorsal intervertebral joints.

The effect of osteo-arthritis of the dorsal joints on the contour of the lumbar column is to accentuate the posterior hollow of the loin, with the result known to clinicians as 'lordosis', as is shown in *Fig.* 591. A result is to throw back the thorax upon the lumbar spine and to bring intervertebral joints under mechanical influences

which properly belong to intervertebral joints of the lumbar curve only. We might call this result 'lumbarization' so far as it affects the thoracic vertebræ. I have no means of proving the surmise, but I should expect that the plumb-line from the skull now falls through a vertebra higher than the normal 12th thoracic.

This process of lumbarization is necessarily progressive once it is initiated. The lumbar curve filches more vertebræ from the thoracic curve and brings them under influences that require them to bear weight by their dorsal joints, and so to develop osteo-arthritis in their turn. Concomitant with this change at the lower end of the thorax is a forward projection of the upper thorax in order to preserve body balance. This change constitutes 'kyphosis', which brings its own immediate problems.

I suggest that the whole outcrop of osteo-arthritis on the dorsal intervertebral joints below the level of the 8th thoracic vertebra is to be interpreted as a first consequence of weight-bearing by the units of the lumbar curve through their dorsal joints and as an extension of the lumbar curve into the thoracic region with the development of lordosis.

THE DORSO-CERVICAL OUTCROP OF OSTEO-ARTHRITIS

The district covered by this outcrop, between the 6th cervical and the 7th thoracic units, should, according to the principles enunciated, be relatively free from osteo-arthritis of the dorsal intervertebral joints. The vertebræ of this region are ordinarily ventriflexed on each other, and vertical strains are met by ligaments rather than by joint surfaces.

An irregular zone of incidence includes the joint between the 7th cervical and the 1st thoracic vertebræ. This high *cervico-thoracic peak*, close to a point of low incidence at the joint next above it, being indeed the maximum point of the whole dorso-cervical outcrop, makes a strong contrast with the even cervical outcrop. The next highest incidence of osteo-arthritis in the dorso-cervical outcrop is at the joint between the 4th and 5th vertebræ—the *upper thoracic peak*.

It will be most convenient to consider first a possible cause of the whole dorso-cervical outcrop, and then the special cause for the *upper thoracic* and the *cervico-thoracic peaks*. The presence of the whole dorso-cervical outcrop is to be sought in factors that may cause dorsiflexion between the vertebræ of this region.

Two vertebral columns I have examined suggest that use of the upper limbs and the connection between them and the thoracic cage may account for the dorso-cervical outcrop as a whole. Much of the upper limb musculature is attached directly to the thoracic cage. The rhomboidei and the trapezius muscles connect the scapula to the thoracic spines. The scapula is strutted to the thorax by the clavicle, and many muscles besides those of the upper arm proper gain attachment to the limb girdle, such as the trapezius and the pectorals. Strong movements of the upper limbs may well imply dorsiflexion of the upper dorsal spine if accompanied by contraction of corresponding parts of the erector spinæ. I might suggest that movements such as those of rowing imply a direct strain from the upper limbs to the upper thoracic spine.

I propose to describe first a column with scoliosis of such character as to suggest the consequences of severe labour performed by the right arm and shoulder. A second column is much less deformed, but is not dissimilar to the first in its essential features. The details of osteo-arthritis of the dorsal intervertebral joints in these

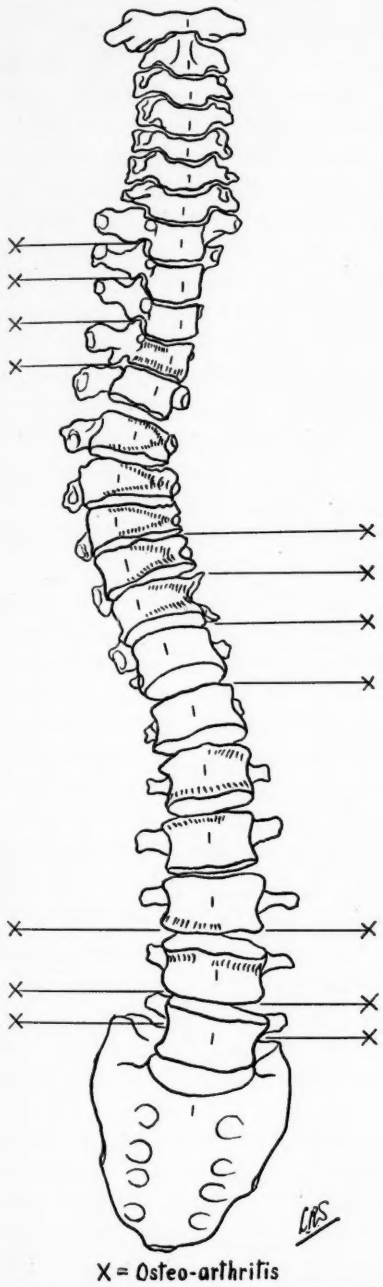

FIG. 592.—Anterior view of a reconstructed column affected with scoliosis. X marks the position in which osteo-arthritis of the dorsal intervertebral joint is to be found. Probably severe labour performed by the right arm and shoulder accounts for the curvature of the upper dorsal spine and for osteo-arthritis in the dorsal joints of that region. Osteo-arthritis is also found in the dorsal joints of the compensatory lower dorsal curvature on the left side. The joint lesions of the lumbar spine are bilateral. The same column is shown in side view in *Fig.* 593.

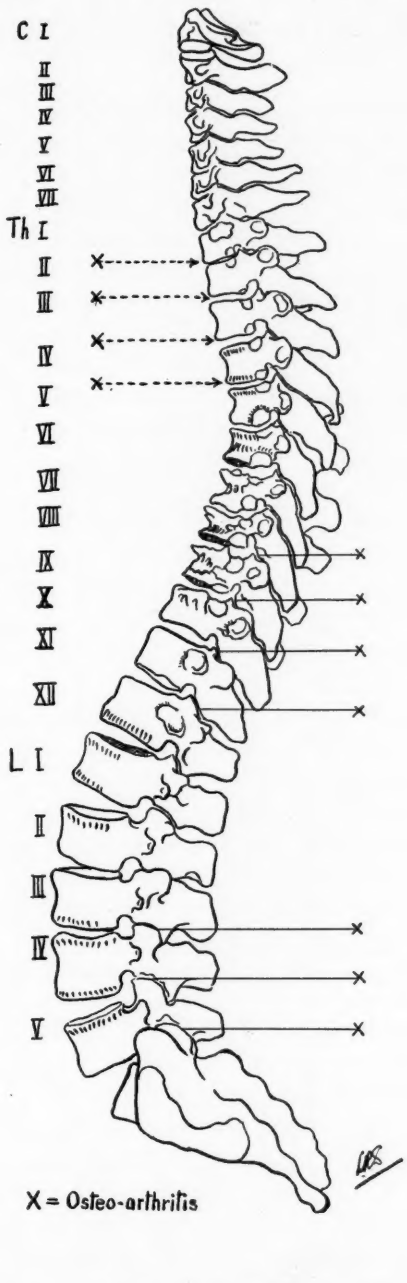

FIG. 593.—Lateral view of the same column as is shown in *Fig.* 592. As before, X indicates osteo-arthritis in the dorsal intervertebral joint. The drawing shows disease in the same positions as *Fig.* 592, and in addition, lordosis developing in the lumbar spine.

two columns may be found to give support to the suggestion that the dorso-cervical outcrop and the use of the upper limbs are associated phenomena.

FIG. 594.—A photograph of a reconstructed vertebral column from the front. Note the curvatures of the spine, which are similar to those shown in *Fig.* 592, though much less pronounced. X indicates osteo-arthritis in the dorsal intervertebral joint in the position indicated.

COLUMN No. I.—Viewed from the front (*Fig.* 592), there appear an upper thoracic curve open to the right; a middle and lower thoracic curve with concavity open to the left; and a slight lumbar curve with concavity open to the right. Viewed from the left (*Fig.* 593), the thoracic curve is much accentuated in its lower members; the cervical curve is somewhat straightened and continues farther than normal into the thorax; the lumbar curve is accentuated and includes the lower two thoracic vertebræ. Rotation of certain vertebral units is shown by the loss of alinement of the vertical marks on the middle anterior points of the vertebral bodies in *Fig.* 592.

In the *upper dorsal curve* osteo-arthritis has affected the joints between the first five vertebræ on the right side only. In the *lower dorsal curve* osteo-arthritis is marked only in the joints from the 8th to the 11th vertebræ on the left, i.e., on the side contralateral to the changes in the upper dorsal region. Osteo-arthritis also affects the costovertebral joints below the 5th thoracic vertebra on the left side. This is probably the result of effort to balance the trunk on the part of the muscles of the anterior abdominal wall, which extend from the lower seven ribs to the pelvis.

In the *lumbar region* osteo-arthritis occurs in the joints between the 3rd lumbar and the lumbo-sacral joint, and is bilateral. Some lordosis is to be inferred, because the vertical line seems to pass through the 10th thoracic vertebra.

This column owes its conspicuous features almost certainly to heavy labour performed by the right arm and shoulder. The contours approximate very nearly to those of the classical specimen described by Arbuthnot Lane to illustrate the effects on the skeleton of heavy labour of the sort performed by the brewer's drayman, who habitually carried heavy barrels on his right shoulder.

In the *upper thoracic region* of this column osteo-arthritic lesions of the intervertebral joints are limited to the right side. In the

complete absence of any gross disease the association of strain transmitted through the right shoulder is too obvious to overlook. Left-sided lesions occur in the left compensatory curve, and bilateral lesions of pure weight-bearing in the lumbar region.

COLUMN NO. II.—This column is shown after reconstruction from a front view in *Fig.* 594. There is curvature of slight degree of the cervical and the upper dorsal spine with the concavity to the right, and of the lower dorsal and the lumbar spine in the opposite direction. Osteo-arthritis of the dorsal intervertebral joints is present in the middle of the cervical curve, between the 1st and 4th thoracic vertebræ and from the 12th thoracic vertebra to the lumbo-sacral joint. In each district the disease is bilateral.

In this column contours are similar to those in Column I, but much less pronounced. Approximately the same district of osteo-arthritis is found in the dorsal intervertebral joints of the upper thoracic region, with the difference that the osteo-arthritic changes are bilateral. I do not think that bilateral distribution invalidates the supposition that osteo-arthritis in the dorsal intervertebral joints of the upper thoracic region is associated in a causal sense with the use of the upper limbs. I should surmise that the individual from whom this second column was derived was not subject to severe asymmetrical labour comparable to that of the brewer's drayman, but was an active man with his hands and right-handed. Only few occupations might furnish examples of excessive use of the right upper limb comparable to the labour of the brewer's drayman; the ordinary activities of labour and of sport demand the use of both arms.

I have specially examined a series of skeletons to determine the side of more frequent occurrence of osteo-arthritis in the dorsal joints between the 7th cervical and the 7th thoracic vertebræ. I find that right-sided lesions constitute 54 per cent of the total, and suggest that this slight preponderance of right-sided disease is associated with the general right-handedness of man. However, the study of the details of Columns I and II suggests to me that the upper dorso-cervical outcrop of osteo-arthritis in the dorsal intervertebral joints is caused by the use of the upper limbs. Such is the provisional explanation I offer for the occurrence.

Frazer[13] remarks that "a slight lateral curve is sometimes seen in the lines of the bodies, concave to the left in the dorsal region and probably due to the use of the right arm, with some compensatory deviation above and below". The columns we have been examining therefore show an exaggerated state of the normal in the matter of their contours.

The Upper Thoracic Peak of the Dorso-cervical Outcrop.—The presumption is that the peak between the 4th and 5th thoracic vertebræ indicates pressure by dorsiflexion in the dorsal intervertebral joints of that district. In this connection we must remember the special function which is attached to the thorax, namely, that of respiration. Halls Dally[14] showed by X-ray examination that straightening of the whole thoracic spine is a normal accompaniment of the inspiratory phase of respiration. The spinous processes are approximated and closely overlap, as Halls Dally suggests, in order to form a natural protection against hyperextension. The vertebral bodies show divergence anteriorly as the intervertebral discs open out and the anterior common ligament becomes taut, and reverse movements accompany the expiratory phase. It must surely follow that the dorsal joints of the thoracic vertebræ become places of pressure with each act of inspiration;

in this case it cannot be a matter for wonder if these joints show the effects of wear and are prone to the development of osteo-arthritis. The normal wear at the part of the thoracic curve that corresponds to the upper thoracic peak might very probably become intensified under certain circumstances.

We have seen how osteo-arthritis in the dorsal joints of the lumbar spine is associated with lordosis, and we have surmised a concomitant kyphosis with reduction of the functional length of the thorax. It is possible that in order to compensate for this reduction of the thoracic spine and of the respiratory tide, the normal excursion of the thoracic spine in dorsiflexion may be increased. Such an event would be expected to throw additional stress upon the dorsal joints.

The concurrence of two circumstances, the use of the upper limb and the respiratory movements, both of which accompany dorsiflexion, may account for the high incidence of osteo-arthritis in the dorsal intervertebral joints at the *upper cervical peak.*

The Cervico-thoracic Peak of the Dorso-cervical Outcrop.—I suggest that the reason why a peculiar strain of dorsiflexion falls on the joints between the 7th cervical and the 1st thoracic vertebræ may lie in the adjustments forced upon the column in order to maintain the poise of the head in the face of alteration of curvature of the spine elsewhere. In the evolution of man perhaps there has been no factor of more constant significance than that of keeping the head erect in order to maintain forward vision. When it is considered what a handicap is placed upon a man if his trunk is bowed forwards, or what a strain is endured if he assume for only the short period of an experiment the attitude of a quadruped, the force of the urge to keep the head upright and poised will be appreciated. No matter how much the trunk is bent, every effort is made to keep the head upright. When the thorax is bowed forwards in kyphosis, then dorsal angulation between neck and thorax must be maintained at whatever level it is necessary to secure the poise of the head.

This surmise may be confirmed from the examination of any series of deformed columns in our museums. In the most deformed columns the neck is commonly found sharply dorsiflexed on the thorax. I suggest that the cervico-thoracic peak of osteo-arthritis may be interpreted in these terms. It is an indication of a compensatory change as the neck forms a new angle with the thorax, and might be described as a result of kyphosis.

THE CERVICAL OUTCROP OF OSTEO-ARTHRITIS

This outcrop of osteo-arthritis extends from the first to the sixth cervical joints, with its maximum at the joint between the 3rd and 4th vertebræ.

The dorsal joints of the vertebræ of the cervical curve carry weight as do those of the lumbar curve, because the vertebral bodies lie in front of the line of gravity. Therefore these joints are specially prone to the development of osteo-arthritis, and as might be expected the point of greatest incidence is near the middle of the curve. Humphry states that the fore part of the 4th vertebra forms the zenith of the curve.

It could be imagined that the uppermost thoracic vertebræ might become functionally absorbed into the cervical curve if lordosis demands a forward projection of the thorax in order to maintain body balance. If this be so, as disease of the dorsal joints spreads the thorax must become functionally shortened, certainly

at the lower end and perhaps at the upper end also. It is remarkable if the adjustments of the trunk to the changing contours of the spine are affected at the expense of the thorax. There is room for research into the change in vital capacity that may accompany lordosis and kyphosis.

However this may be, it is difficult to explain the very high incidence of osteo-arthritis at the cervico-thoracic junction as an extension of the cervical curve. A special local function explaining that occurrence has already been discussed.

OSTEO-ARTHRITIS OF THE ANTERIOR ATLANTO-AXOID JOINT

Out of all the columns showing osteo-arthritis I examined, 32·2 per cent of specimens of the atlas and axis showed disease in this joint. Comparison of this figure with the graph in *Fig.* 590 will show that osteo-arthritis is commoner in the anterior atlanto-axoid joint than in any of the dorsal intervertebral joints, except in those of the lumbar region.

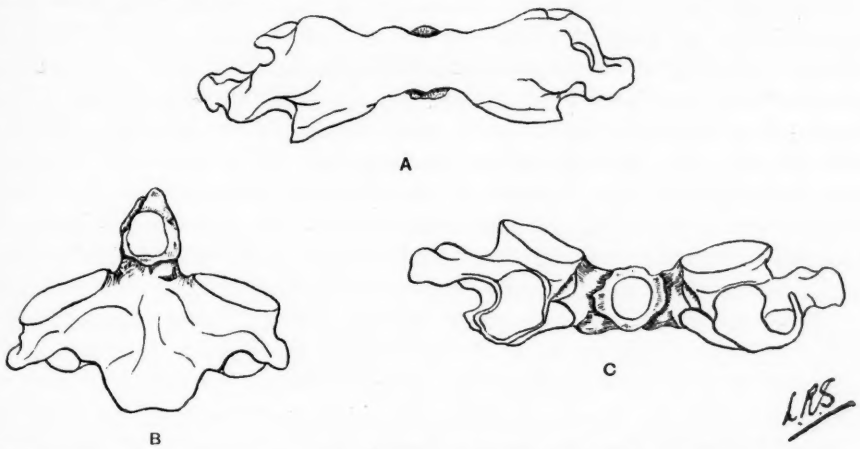

FIG. 595.—Drawings showing: A, The anterior arch of the atlas from the front with projecting osteophytes in the middle; B, The front of the axis with odontoid process; and C, The anterior arch of the axis from the back after the posterior arch has been removed. B and C show the two contact surfaces of the anterior atlanto-axoid joint; note peripheral osteophytes indicating early osteo-arthritis (*Stage I*).

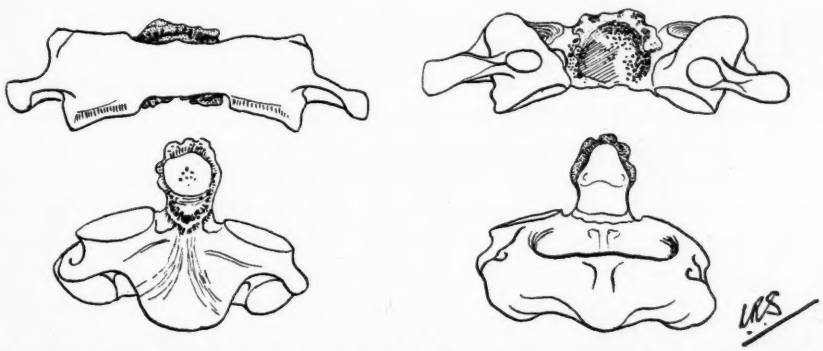

FIG. 596.—Drawings of a specimen of the atlas and of the axis vertebra showing osteo-arthritis of the anterior atlanto-axoid joint of a more advanced degree (*Stage III*). The two upper drawings show the anterior arch of the atlas from the front (left) and the back (right). The two lower drawings show the axis and the odontoid process from the front (left) and the back (right). Cross-hatching in the upper right drawing indicates eburnation.

The contact surfaces of this joint are the front of the odontoid process of the axis and the back of the anterior arch of the atlas, and the movement in this joint is universally conceded to be rotatory. It is remarkable that Adams, to whom reference has already been made, gives to disease of this joint a very large share of his account of osteo-arthritis of the small synovial joints of the vertebral column. He wrote[15]: ". . . the atlas and the dentata have far more free movements on each other than any of the other vertebræ enjoy, a circumstance that may account for the observation that we are accustomed to see far more numerous specimens of the effects of chronic rheumatic arthritis in these vertebræ than in all the others of the spine taken collectively". My findings do not confirm this final statement, but certainly osteo-arthritis of the anterior atlanto-axoid joint is very common.

Drawings showing disease in this joint will be found in *Figs.* 595 and 596. Arbuthnot Lane notes osteo-arthritis ('rheumatoid arthritis') of this joint in his account of labour changes and the effects of weight-carrying. It is described in connection with the occupation of the coal-heaver, who carries weight on the back supported by the head and neck through the medium of a long-flapped cap.

Osteo-arthritis of the anterior atlanto-axoid joint might well have a clinical interest. In this position it might very well be the cause of occipital headache and of stiff neck if Hilton's[16] well-known generalization is accepted: "The same trunks of nerves, whose branches supply the groups of muscles moving a joint, furnish also a distribution of nerves to the skin over the insertions of the muscles; and . . . the interior of the joint receives its nerves from the same source". Probably the anterior atlanto-axoid joint receives its nerve-supply from the first cervical nerve, which lies very close to the joint and supplies the short rotator and extensor muscles of the suboccipital space and the semispinalis capitis of the nuchal region also. The skin does not receive a direct supply from the first cervical nerve, but by reason of a communication with the second cervical, the first cervical nerve takes a share in the supply of an area of skin to which the great occipital nerve is distributed. Therefore, an area of skin extending from the vertex to the nuchal crest and from mastoid process to mastoid process might be the seat of sensation referred from the anterior atlanto-axoid joint by the first cervical nerve.

SUMMARY AND CONCLUSIONS

This paper is devoted to the consideration of osteo-arthritis of the small synovial joints of the vertebral column. This condition is to be distinguished from 'osteo-arthritis of the spine'. In a description of the pathological anatomy of osteo-arthritis three stages of development are described. In the first the disease is indicated only by a fringe of osteophytes around the normal contact area; in the second by a zone of porous bone which separates the original contact area from a peripheral fringe of osteophytes; in the third stage all traces of the original contact area are lost and the surface may be grooved, polished, and greatly deformed.

The distribution of osteo-arthritis in the dorsal intervertebral joints, which has not previously been recorded, is set out in graphic form, and certain zones of special incidence are observed.

1. **Lumbo-dorsal Outcrop.**—This is interpreted as the result of weight-bearing in the joints of the dorsiflexed lumbar column, and the absorption of the lower thoracic vertebræ into the lumbar curve as lordosis is established.

2. **Dorso-cervical Outcrop.**—It is suggested that in the main this outcrop is the result of the use of the upper limbs, by movements of dorsiflexion transferred from the limbs to the thoracic skeleton and by associated action of the erector spinæ muscle. This outcrop presents two peaks of higher incidence :—

a. At the joint between the 4th and 5th thoracic vertebræ is found the *upper thoracic peak.* It is suggested that this peak is due to dorsiflexion which is a normal feature of inspiration accentuated with the onset of lordosis and kyphosis.

b. The cervico-dorsal junction is marked by a *cervico-dorsal peak.* It is suggested that the explanation lies in the powerful urge to keep the head upright in spite of kyphotic changes in the thorax. Columns greatly deformed with kyphosis often show that the head and neck are borne upright in spite of great postural difficulties.

3. **Cervical Outcrop.**—This is probably due to weight bearing in the joints of the already dorsiflexed cervical vertebræ.

Some space is given to a description of osteo-arthritis in the anterior atlanto-axoid joint. Disease in this position was found in about one-third of columns which exhibit osteo-arthritis elsewhere in the dorsal intervertebral joints.

I have pleasure in making grateful acknowledgements to those who have assisted me by providing facilities for observation, or by other means. Professor R. A. Dart, of the University of the Witwatersrand, allowed me full access to the collection of skeletons in the Anatomical Museum. It was here that the first observations were made. My investigations were continued at Cambridge, where Professor J. T. Wilson and Dr. W. L. H. Duckworth gave me full opportunity of examining the skeletons in the Anatomy Department. Sir Walter Langdon-Brown, Regius Professor of Physic at Cambridge, and the M.D. Committee permit me to publish these observations, which formed part of a thesis presented for the degree of M.D. in June, 1933.

REFERENCES

[1] ADAMS, ROBERT, *A Treatise on Chronic Rheumatic Arthritis*, 1857, 283. London.
[2] LANE, W. A., "Some Points in the Physiology and Pathology of the Changes produced by Pressure in the Bony Skeleton of the Trunk and Shoulder Girdle", *Guy's Hosp. Rep.*, 1886, xliii, 321.
[3] FISHER, TIMBRELL, *Chronic Non-tuberculous Arthritis*, 1929, 118.
[4] LANE, W. A., "The Causation and Pathology of the So-called Disease, Rheumatoid Arthritis, and of Senile Changes", *Trans. Pathol. Soc. Lond.*, 1886, xxxvi, 387.
[5] FISHER, TIMBRELL, "A Contribution to the Pathology and Etiology of Osteo-arthritis", *Brit. Jour. Surg.*, 1922, x, 73.
[6] LEGG, WICKHAM, "A Second Case of Hæmophilia with Examination of the Tissues and Joints", *Trans. Pathol. Soc. Lond.*, 1885, xxxvi, 412.
[7] HASTINGS, GIFFORD, "On a Condition of Mixed Premature and Immature Development", *Med.-Chir. Trans.* 1897, lxxx, 17.
[8] GARROD, SIR ARCHIBALD E., *Proc. Roy. Soc. Med.*, 1923, xvii, 2.
[9] FISHER, TIMBRELL, *Loc. cit.* (3).
[10] FISHER, TIMBRELL, *Loc. cit.* (3), 50.
[11] HUMPHRY, G., *The Human Skeleton*, 1858, 147.
[12] SHORE, L. R., "A Report on the Nature of Certain Bony Spurs arising from the Dorsal Arches of the Thoracic Vertebræ", *Jour. of Anat.*, 1931, lxv, 379.
[13] FRAZER, J. E., *The Anatomy of the Human Skeleton*, 1914, 14.
[14] DALLY, HALLS, 1908, "The Movements of Respiration", *Jour. of Anat. and Physiol.*, 1908, xliii, 98.
[15] ADAMS, ROBERT, *Loc. cit.*, 287.
[16] HILTON, JOHN, *Rest and Pain*, 1880, 3rd ed., 168.

POLYSPONDYLITIS MARGINALIS OSTEOPHYTICA

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INTRODUCTION

A COMMON disease can be recognized in *Fig.* 597, which is the photograph of a vertebral column reconstructed from its constituent bones. The drawing and the radiogram reproduced in *Fig.* 598 will perhaps also recall the same familiar condition. So far as I can find, this disease, though probably the commonest to which the vertebral column of man is subject, has no precise name. 'Spondylitis' alone—or perhaps with the epithet 'hypertrophic'—'spondylitis deformans', 'spondylosis', and 'osteo-arthritis of the spine' probably include most of the synonyms in common use.

No definite accounts are to be found in standard works on pathology, medicine, or surgery of the anatomy of these osteophytes or of their distribution in the vertebral column. The observations on these points recorded in this paper were made upon vertebral columns which had been macerated, dried, and rendered free from putrescible matter. Enough fresh material was obtained from the post-mortem room to make examination of the soft tissues possible in addition.

GENERAL DESCRIPTION

The vertebral column shown in *Fig.* 597 presents features familiar to every museum student; and the features of the specimen shown in *Fig.* 598 are equally familiar to the radiologist or the pathologist. A knife thrust into an intervertebral interval of such specimens would meet bone. This event is to be expected from the findings by radiologists of fringes, of ledges, and of other bony projections from the upper and the lower edges of vertebral bodies which constitute 'lipping'.

The bony growths from macerated vertebræ have the following superficial characters:—

1. Osteophytes commonly arise as spicules or as fringes of spicules from adjacent margins of vertebral bodies on their anterior or lateral aspects. I have never seen a vertebra bear osteophytes on the edge that is posterior and abuts on the spinal canal, and Beadle[1] makes the same observation.

2. Osteophytes usually bear longitudinal striations of ridges and grooves, best seen in osteophytes of moderate size.

3. Osteophytes are always broader at the base than at the point and spread over the anterior and the lateral surfaces, but not over the horizontal surfaces of the vertebral bodies.

4. Osteophytes may be larger, less diffuse, and nodular; and these may span an intervertebral interval. The outer surfaces of such nodules may be smooth or with striation retained at their bases only. Hemispherical, discoidal, or pyramidal forms may occur, and bizarre shapes also if adjacent nodules make contact.

5. Almost never are osteophytes to be found on a single unit of the vertebral column. Their incidence is spread over a group or over more than one group of adjacent vertebræ.

NOMENCLATURE

Chronic disease of the vertebral column has long received the name 'spondylitis deformans' when permanent changes in the bones and ligaments are associated with lasting alteration of posture—usually a kyphotic curve of the spine.

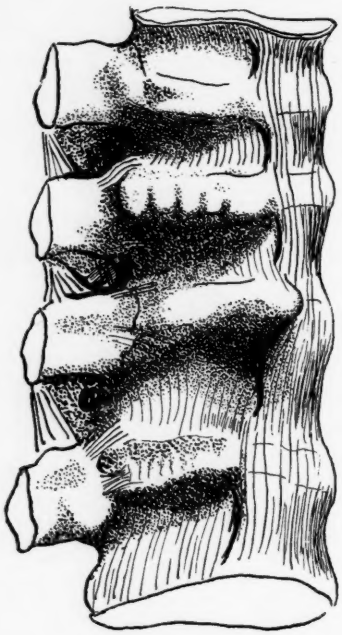
'Spondylitis', derived from σπόνδυλος, a vertebra, strictly refers to a unit of the vertebral column. The whole column, in its primitive form the ridged backbone of a quadruped, is ῥάχις, whence is derived 'rachitis'. It would be misleading and pedantic to insist on the use of the term 'tuberculous spondylitis', though it would be more usually correct in both the pathological and the etymological sense. A disease spread widely over the vertebræ would be better rendered by 'rachitis' were not the word used as equivalent of 'rickets'. 'Senile rachitis' suggests a paradox; so perhaps 'polyspondylitis', indicating a plural incidence of disease in the vertebræ, is to be preferred to the singular form 'spondylitis'.

FIG. 597.—Photograph of a vertebral column affected with polyspondylitis marginalis osteophytica. The presence of osteophytes growing from the upper and lower margins of the vertebral bodies is clearly shown. It is also to be noted that the distribution of these osteophytes corresponds very nearly to that indicated in *Fig.* 601. Outcrops of osteophytes are to be seen in the cervical, the thoracic, and the lumbar regions, but osteophytes are smaller or absent in the vicinity of the 2nd thoracic, the 12th thoracic, and the lower margin of the 5th lumbar vertebræ.

Many authors use 'spondylitis deformans' to cover an imperfectly defined group of diseases which are all marked by new bone formation in the vertebral column. Usually 'spondylitis deformans' is described in the chapter or section devoted to 'osteo-arthritis'. Again, some authors use the term 'osteo-arthritis of the spine' to describe the disease which is the subject of the present note. The mutual growth of osteophytes from the bony elements of an intervertebral joint suggests a similarity to osteo-arthritis, which also is marked by bony growths from the margins of joints.

In common use 'osteo-arthritis' refers to a chronic disease of the so-called 'diarthrodial'

or 'synovial' joints, which are distinguished in every case by a cavity, by cartilage-covered contact surfaces, by a synovial membrane, and by a fibrous capsule. The joint between a pair of vertebral bodies has neither cavity nor synovial membrane. The flat ends of the vertebral bodies are partly covered by cartilage plates on which are built the intervertebral discs. These discs are composed of concentric plates of fibrous tissue which form a shell (annulus fibrosus) surrounding in the centre a mass of mucin-like material (nucleus pulposus) which is elastic, expansive, and retained under pressure. It is not to be expected that such a joint is subject to the same diseases as a synovial joint. Timbrell Fisher[2] has showed that true osteo-arthritis may be produced in the laboratory animal by methods which are quite inapplicable to a joint of any type but synovial.



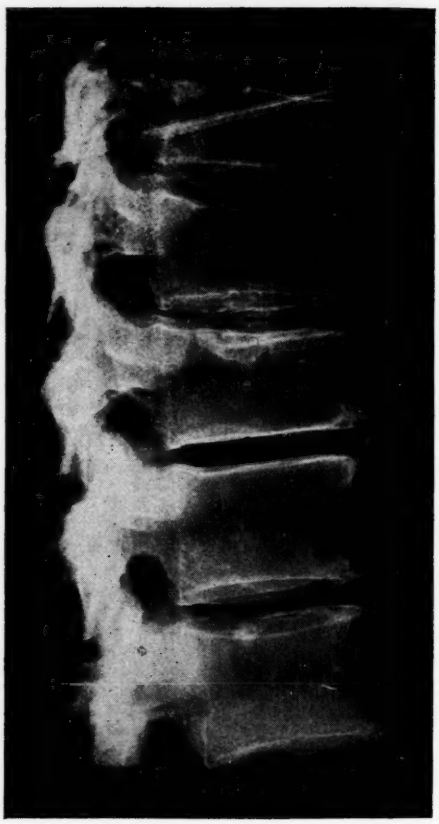


FIG. 598.—On the left is a drawing from a photograph of the middle part of the thoracic region of a diseased column. The specimen is viewed from the right anterior aspect, and the thickenings at the intervertebral intervals due to marginal osteophytes gives a 'moniliform' appearance. There is less bulging in the region covered by the anterior longitudinal ligament, which is clearly indicated in the drawing. The bulging lies between the edge of that ligament and the back of the vertebral bodies in the region of the costo-vertebral joints. In the middle of the specimen a bony nodule projects forwards and medially to overlap the front of the anterior longitudinal ligament. On the right is a reproduction of a radiogram of the same specimen taken in a lateral position. 'Lipping' of the vertebral margins is clearly shown, especially at the prominent nodule in the middle of the specimen.

The osteophytes of osteo-arthritic joints, as seen in macerated bones, are the remains of cartilage-capped chondrosteophytes grown from the articular margins. In polyspondylitis marginalis osteophytica the osteophytes are osseous substitutions for ligament, and this probably accounts for the not uncommon occurrence of ankylosis, which is very rare in true osteo-arthritis.

Lawford Knaggs makes a welcome break with tradition when he gives a separate chapter to spondylitis deformans in his *Inflammatory and Toxic Diseases of Bone*. He distinguishes the disease whose features bear superficial resemblance to osteo-arthritis as ' spondylitis osteo-arthritica ' from among other diseases peculiar to the vertebral column. This disease I confidently identify with polyspondylitis marginalis osteophytica, though Knaggs offers no exact description of its anatomy or distribution.

It is with much diffidence that one advocates the introduction of a fresh name into the vocabulary of pathology, especially when the name refers to a very common disease. ' Polyspondylitis ' has been explained, and the justification of ' marginalis ' will appear.

ANATOMY

1. Marginal osteophytes are not related to the attachments of muscles or of tendons. Though large nodules are found in the lumbar spine, and though fibres of the psoas muscle are attached alike to the ligaments which cover the nodules

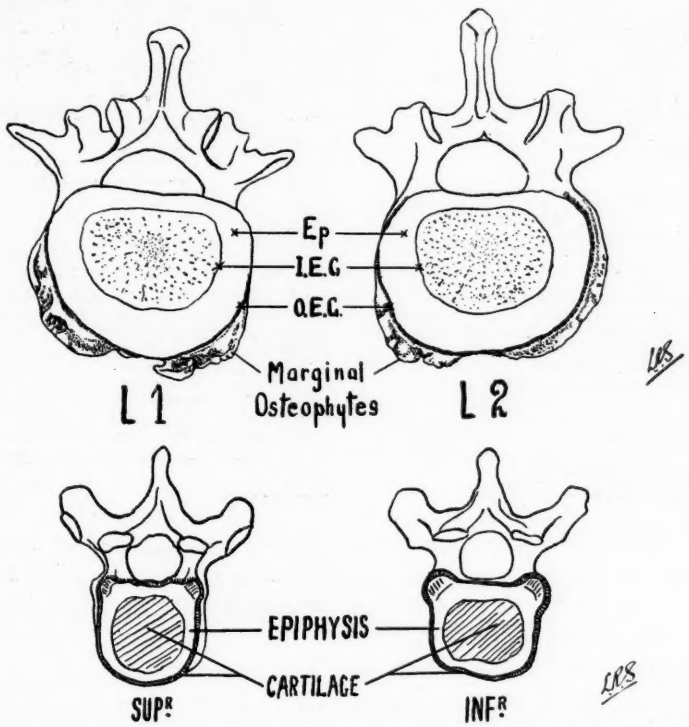


FIG. 599.—The two upper drawings show on the left the lower surface of the 1st and on the right the upper surface of the 2nd lumbar vertebra of the same column. Ep, Epiphysial ring; I.E.G., Inner epiphysial groove. The outer epiphysial groove (O.E.G.) becomes conspicuous in the presence of marginal osteophytes. The two lower drawings show the proportions of the epiphyses of the vertebral bodies in earlier life. The specimen, drawn both from above and below, is a thoracic vertebra from the skeleton of a male aged 16 years. The epiphyses, which bear the facets for the costal articulations, are embedded in cartilage. This cartilage appears between the edge of the epiphysis and that of the vertebral body, and also in the central part of the flat surface of the vertebral body.

and the normal parts of the lumbar spine, I have never observed an isolated tendon to be attached to a nodule. The same statement may be made of the muscle longus colli, though it must be admitted that nodules are rare in the cervical spine.

2. The osteophytes arise or ' break the surface ' at a position on the vertebral body which is quite constant. If an affected vertebra is examined from the upper or the lower surface, it can be seen that the osteophytes are marked off from the body of the vertebra that bears them by a groove which marks their inner limits. It is by this arrangement that the word ' marginal ' is justified. In *Fig.* 599, which presents drawings of the flat surfaces of osteophyte-bearing vertebræ, these grooves are indicated, O.E.G. representing ' outer epiphysial groove '.

The upper and the lower surfaces of the vertebral body from any part of the column bear a circumferential ring of slightly raised, flat, smooth bone. The inner edge of this ring is often irregular but contains an area which is porous in the macerated bone. In the fresh state this area is covered by a cartilage plate which closes the cancellous spaces of the vertebral body and abuts on the intervertebral disc. In early life this bony ring, which is the epiphysis of the vertebral body, is set in cartilage and falls short of the edge. At full growth the cartilage is ossified further and the ring reaches the edge. The inner edge of the ring is clearly defined

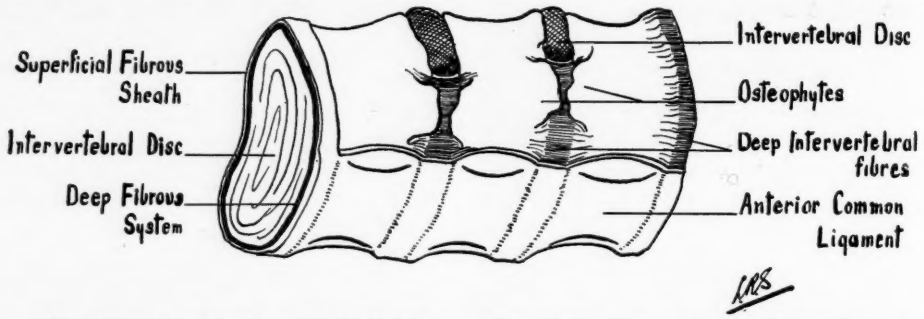


FIG. 600.—Shows in diagrammatic fashion the mode of formation of marginal osteophytes. A portion of a vertebral column is viewed from the lateral aspect. From the sides of the column the superficial lateral expansions of the anterior longitudinal ligament have been dissected away, but the front of the anterior longitudinal ligament is left intact. On the left a section has been made through the intervertebral interval and shows (*a*) the intervertebral disc with its sheath, (*b*) the deep intervertebral ligaments cut across, and (*c*) the superficial fibrous system left as a cuff surrounding the column in the immediate vicinity of the section. After removal of the superficial fibrous sheath the deep intervertebral ligaments are displayed. On the right of the drawing these ligaments are shown in continuous series. In the other intervertebral intervals osteophytes are shown invading and replacing the deep intervertebral ligaments. Towards the top of the drawing are represented the sheaths of the intervertebral discs which come into view after removal of the deep fibres.

(NOTE.—Anterior common ligament = anterior longitudinal ligament.)

and may be called the ' inner epiphysial groove '. Occasionally the outer edge of this ring may be traced in an adult bone as a very fine and narrow ' outer epiphysial groove '. It is when the osteophytes develop that the outer epiphysial groove becomes conspicuous, for a reason which will appear. The outer epiphysial groove receives the attachment of the sheath of the intervertebral disc.

3. *The Perivertebral and the Intervertebral Ligaments.*—The whole vertebral column is enveloped in a continuous sheath of fibrous tissue. This conception, put forward by Macalister and confirmed by Poirier and Charpy, will be shown to be of some importance. The perivertebral sheath is weak at the sides, but medially, both in front and behind, specializes into anterior and posterior longitudinal ligaments. The fibres of these two ligaments are long, and pass over as many as three or four vertebræ. Deep fibres which span no more than one intervertebral interval lie between the intervertebral discs and the more superficial fibres.

These deep fibres are attached very firmly to the edges of the vertebral bodies. It is in these short deep intervertebral ligaments adjacent to the discs and outside

the outer epiphysial groove that the osteophytes of polyspondylitis marginalis osteophytica arise, and it is by the spread of an ossific process into them that the outer epiphysial groove is made conspicuous.

4. The superficial sheath of the vertebral column, of which the anterior common ligament is a component part, can be dissected away from the osteophytes quite readily, in spite of the partial blending of the fibres of the deep and the superficial systems. This point needs some emphasis because it has been claimed by Beadle that the longitudinal striations of the osteophytes " represent the fibres of the anterior common ligament, which over a long time has undergone a severe stretching force ". In addition to the reason given above, it may be remarked that the osteophytes most commonly arise in a part of the circuit of the vertebral body which is lateral to the anterior common ligament; an example is shown in *Fig.* 598.

Fig. 600 shows the mode of formation of the osteophytes in schematic fashion. The drawing, based on the finding of a number of dissections, shows on the left a section made through an intervertebral interval. The ligaments, superficial and deep, are left as a cuff round the intervertebral disc. For the rest of the drawing, the superficial ligaments have been removed in a longitudinal strip and osteophytes are revealed lying in a deeper stratum, being embedded in the deep intervertebral fibres. In their turn these deep fibres have been removed in places to show the intervertebral discs and their sheaths.

DISTRIBUTION OF OSTEOPHYTES

The distribution of the osteophytes, determined from observations made on 106 vertebral columns, is shown in graphic form in *Fig.* 601. In collecting the data on which this graph is based the vertebræ were noted which bore characteristic osteophytes, and also the margins—upper, lower, or both—from which the osteophytes projected. The final count revealed inequalities in the total numbers of vertebræ, due to loss or damage—for complete vertebral columns in a condition fit for examination are not too readily come by—and therefore the results are presented in percentage terms. In the graph the osteophyte-bearing capacity of each vertebral margin is shown as if the total were 100.

The graph makes clear the following points in distribution :—

1. The liability of vertebræ to bear marginal osteophytes increases as the column is traced downwards.

2. There are three well-defined ' outcrops ': (*a*) The *cervical outcrop* is marked off from the thoracic by a minimum zone at the junction of the 1st and 2nd thoracic units. (*b*) The *thoracic outcrop* is higher than the cervical, and is separated from the lumbar outcrop by a diminution zone at the 12th thoracic vertebra. (*c*) The *lumbar outcrop* is higher than the thoracic, but falls to a minimum at the lumbosacral junction. These three outcrops and minimum zones are shown in *Fig.* 597.

3. The osteophyte-bearing capacity of a pair of vertebral margins separated by the same intervertebral disc is not always the same. For example, the graph shows that more marginal osteophytes occur at the upper margin of the 2nd than at the lower margin of the 1st lumbar vertebra.

Commentary on Distribution.—The zones of diminution show that weight in itself cannot explain the occurrence of marginal osteophytes. The weight of the trunk must increase from the 2nd to the 12th thoracic vertebræ and thence to the lumbo-sacral junction, but it is at these places that incidence is reduced.

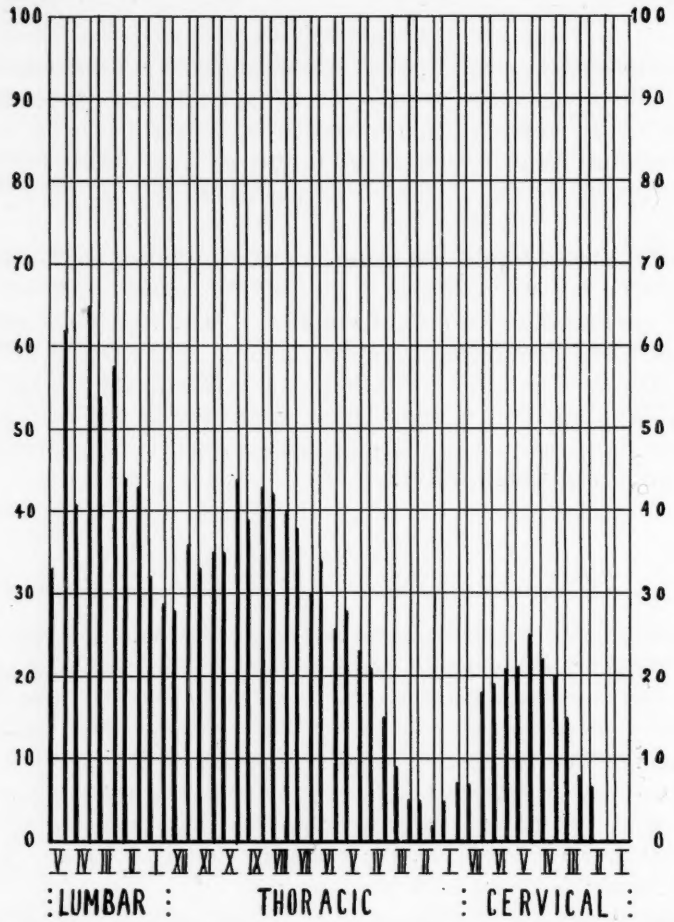


FIG. 601.—Graph showing the distribution of the osteophytes of polyspondylitis marginalis osteophytica in the human vertebral column. The abscissæ represent the several vertebræ set out in ascending order from left to right. Above the abscissæ are constructed upright rectangles whose right and left sides respectively represent the upper and the lower margins of the different vertebræ. Ordinates in heavy black on the sides of these rectangles indicate the numbers per centum of the upper or of the lower margins of the several vertebral bodies which have been found to bear osteophytes. The three-waved form of the graph is to be especially noted, and the positions of the minimum points—at the 2nd and 12th thoracic vertebræ and at the lower edge of the 5th lumbar vertebra. It will be observed that each wave increases in size as the column is traced downwards.

No author has described the curves of the vertebral column more carefully than has Humphry in *The Human Skeleton*. I have had the opportunity of examining Humphry's original preparations preserved at Cambridge and accept his conclusions *in toto*.

Humphry found that the mobile units of the vertebral column are arranged in three alternating antero-posterior curves, and that the junction points of these curves lie in the same vertical line with the line of gravity of the head and of the heads

of the femurs. The reader may compare the points and regions of the vertebral column defined by Humphry with the significant points and regions of incidence of marginal osteophytes in the table set out below.

Anatomical Observation (*Humphry*)	Significance	Marginal Osteophytes
Tip of odontoid process	= top of cervical curve	= zero
4th cervical vertebra	= zenith of cervical curve	= cervical maximum
2nd thoracic vertebra	= bottom of cervical and top of thoracic curve	= *diminution zone*
7th or 8th thoracic vertebra	= zenith of thoracic curve	= thoracic maximum, 7th to 10th vertebra
12th thoracic vertebra	= bottom of thoracic and top of lumbar curve	= *diminution zone*
	= ? zenith of lumbar curve	= lumbar maximum, 3rd to 5th vertebra
5th lumbar vertebra	= bottom of lumbar curve	= *diminution zone*

(The zenith of the lumbar curve is not stated by Humphry, who remarks that the curve is not the arc of a circle.)

Evidently the explanation of the remarkable distribution of marginal osteophytes is to be found in the architecture of the vertebral column. The diminution zones are at the balanced vertebræ through which the weight of the trunk falls vertically. These vertebræ mark points of relative stasis in relation to the posture and the ordinary movements of erect-standing man. For each curve, except the lumbar, the point of maximum incidence and the zenith have a near correspondence.

CAUSATION

It has been shown that marginal osteophytes result from an ossific process in the deep intervertebral ligaments, and that it is the configuration of the vertebral column that determines the points of greatest and of least incidence.

That pressure is a prime causative factor is shown by the changes that occur in the edges of a vertebra situated in the concavity of a scoliotic spine. Vertebræ in such a situation become wedge-shaped, and on the side where compression is greatest develop flanges which have all the characters of marginal osteophytes. In such vertebræ, however, the outer epiphysial grooves become obscured early. Such a column as that shown in *Fig.* 604 results from some specialized attitude which has disturbed the whole balance of the column. Apart from such an extreme, evidence of the predominant effect of mechanical influences is to be inferred from the distribution.

Section of vertebral bodies with marginal osteophytes shows that the usual change is in the direction of increased density, and collapse of the bodies is rare. It is to be deduced, therefore, that disease of the vertebral bodies is not the starting-point. Evidence may, however, be adduced to suggest that the primary cause may be in the intervertebral discs. The normal functions of an intervertebral disc include the maintenance of an expansive turgescent nucleus between each pair of vertebral bodies, to form a pivot for such rotation as vertebral bodies are capable of and to constitute about one-fourth of the height of the vertebral column.

Independent research by Ross-Smith[3] and by Beadle has proved that degenerative change in the nucleus of the intervertebral discs follows the fourth decade of life and is progressive thereafter. An intervertebral disc without its turgescent nucleus, instead of keeping a pair of vertebral bodies in proper separation, is no

more than a mass of inelastic tissue to be pressed and moulded at the direction of the vertebræ between which it lies. It is possible that degenerated discs will spread under weight and exert pressure on circumferential ligaments by bulging them. Or vertebral bodies might slide or rotate on their neighbours and so throw strain on the intervertebral ligaments. That bulging of the intervertebral discs and of the overlying ligaments does occur is readily shown. *Fig.* 602 shows tracings of vertical sections through the same specimen as that illustrated in *Fig.* 598. Further sections with similar findings are shown in *Fig.* 607.

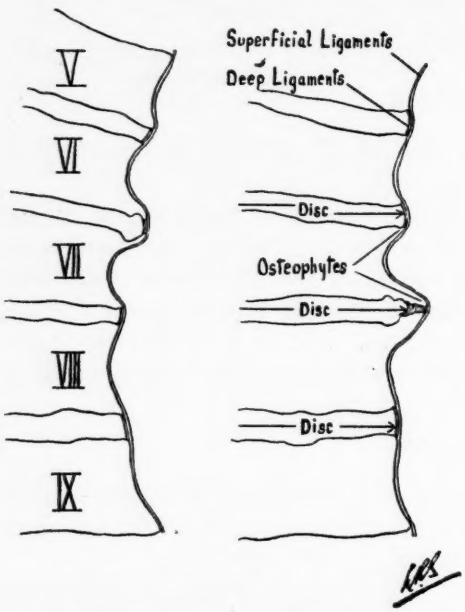


FIG. 602.—Tracings of sections made through the same specimen as is shown in *Fig.* 598. In order to show the bulging of the intervertebral discs that underlie the osteophytes, the specimen was cut with a fretsaw in two vertical planes. In each section the superficial ligaments that form the perivertebral fibrous investment, the deep intervertebral ligaments, and the outer limits of the intervertebral discs are shown in diagrammatic fashion. The tracing on the right traverses the conspicuous nodule shown in *Fig.* 598. It will be seen that the intervertebral disc is bulged, the outer epiphysial grooves are conspicuous, and the osteophytes are embedded in ligamentous tissue. A very small osteophyte is shown at the upper edge of the 7th vertebra, also accompanied by a slight bulging of the disc. The tracing on the left is in a plane slightly to the right of the other. Early osteophytes are shown growing from adjacent margins of the 6th and 7th vertebræ around the bulging intervertebral disc.

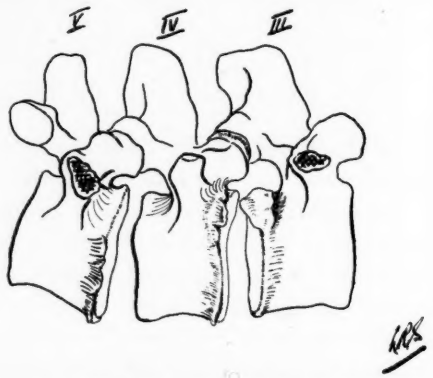


FIG. 603.—The three lumbar vertebræ in this drawing show an early stage in the growth of osteophytes from their margins. Distinct osteophytic flanges have begun to grow from the upper margins of the 4th and 5th lumbar vertebræ, while the lower margin of the 3rd and 4th are free. This mode of growth is usual in the lumbar column. The foramen between the 3rd and 4th vertebræ is partly obstructed by osteophytes. Growth of osteophytes in this position is uncommon, but might well be expected to cause a lesion of the 3rd lumbar nerve at its exit from the vertebral canal.

Sliding of vertebral bodies seems the most probable explanation of the phenomenon briefly referred to earlier, that osteophytic growth in the lumbar vertebræ is more extensive on the lower of an adjacent pair of margins than on the upper. The graph in *Fig.* 601 and the lumbar vertebræ shown in *Fig.* 603 illustrate this occurrence. This arrangement seems to be the outcome of the contours of the cervical and the lumbar curves, in which the vertebral bodies lie in front of the line of gravity and weight bearing. In the thoracic curve, in which the vertebral bodies lie behind the 'plumb-line,' the same arrangement is found as high as the 9th vertebra or so; above that level the arrangement may be reversed.

In the lumbar curve, in consequence of the contour of the curve and the slope of the vertebral bodies, the weight of the column may thrust the disc forwards. This second factor, which is additional to the general bulging, affects only the

lower attachments of the ligaments and may account for the greater ossification in those positions.

The longer anterior and posterior ligaments probably do no more than limit extreme dorsi- and ventri-flexion. Rotatory movements of vertebral body on vertebral body are limited by the deep intervertebral ligaments. Evidence that rotation does take place between vertebral bodies and is associated with the growth

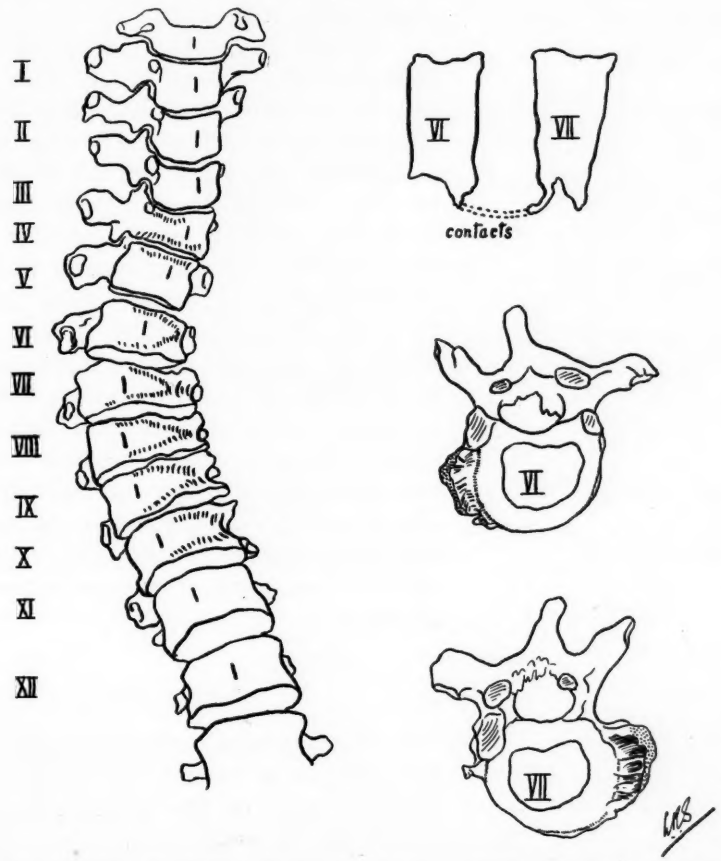


FIG. 604.—Drawings of certain specimens derived from a column which was the subject of scoliosis. The thoracic region, which was principally affected, is shown on the left in front view after reconstruction. The anterior mid-points of the vertebral bodies, determined by measurement, are shown by vertical lines. Evidently twisting has taken place between certain vertebræ, e.g., between the 3rd and 4th and between the 6th and 7th vertebræ. On the right above are shown the appearances of the 6th and 7th vertebræ in coronal section—note the wedge deformity of the 7th and the flangeing of both vertebræ. Below are drawings of the adjacent surfaces of the same two vertebræ. The osteophytic flanges arise outside the outer epiphysial grooves in both specimens. Growth is evidently greater on the upper aspect of the 7th vertebra, in which the outer epiphysial groove has become partly obliterated. The osteophytic growths from the 6th and 7th vertebræ made contact by areas which are distinguished by stippling in the two lower right-hand drawings.

of marginal osteophytes is not difficult to obtain. In *Figs.* 604 and 605 the anterior mid-points of the vertebral bodies, determined by measurement, are marked by short vertical lines and make lack of alinement obvious. In the column shown in *Fig.* 605 ankylosis has taken place between two vertebræ, and the rotation must have preceded ankylosis. Similar results are often obtained by measurement and reconstruction.

When the vertebral column begins to ' give ' under weight, one curve may very probably rotate on the next and throw a strain on the deep intervertebral ligaments. The ' anticlinal ' vertebræ lie in the axis of rotation and suffer least from this effect. The further from the point of balance and the nearer the zenith of a curve, the greater is the excursion and the more intense is the strain in the ligaments.

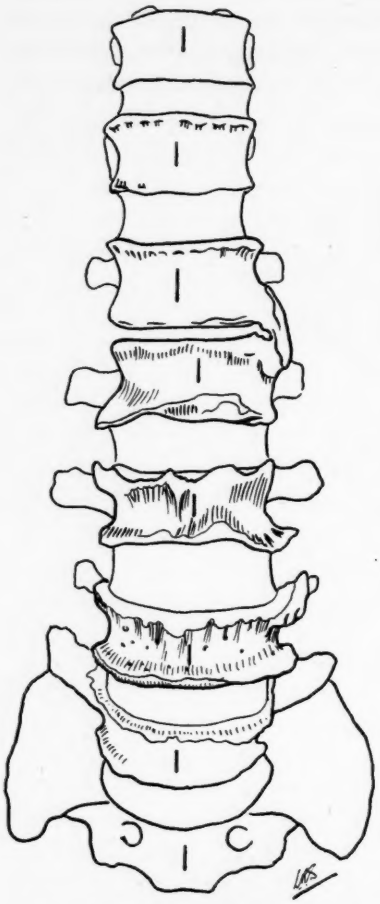


FIG. 605.—Anterior tracing of part of a reconstructed vertebral column showing the rotation that has taken place between vertebra and vertebra. The anterior middle points of the vertebræ, determined by measurement, are indicated by black vertical lines. Ankylosis has taken place between the 1st and 2nd lumbar vertebræ on the left side ; lack of alinement of the middle points shows that twisting must have preceded ankylosis.

A survey of the sheath of superficial ligaments that surrounds the deep ligaments and the intervertebral discs may explain the different degrees of osteophyte growth found in different parts of the circuit of the vertebral column. Osteophytes are uncommon on the front and unknown on the back of the vertebral margins, where the anterior and posterior longitudinal ligaments lie thick and strong. Laterally, the perivertebral fibrous sheath, of which the two long median ligaments are local thickenings, are thin and ineffective as supports to deeper structures. It is in the arc of the circuit of the vertebral margins between the two long ligaments that the majority of osteophytes arise. *Fig.* 598 shows that osteophytes may arise laterally to the anterior longitudinal ligament and grow to overlap the front of the ligament.

ANKYLOSIS

Many authors comment on the proneness to ankylosis in ' osteo-arthritis of the spine ', and the rarity of the event in osteo-arthritis of ordinary synovial joints.

Fig. 606 is a drawing of part of a vertebral column, and shows how osteophytes grow to meet each other from vertebra to vertebra in a strangely purposive manner. This need surprise us the less when we realize that ossific substitution for ligament is taking place from each of its opposite attachments. Osteophytes, either by interlocking or by bony ankylosis, may form a splint and may set a limit to disease which is otherwise progressive, and may bring about spontaneous cure in which rigidity is effected at the cost of mobility.

This tendency to ankylosis has been exaggerated. Undoubtedly ankylosis takes place and commonly enough ; but more often osteophytes with every superficial appearance of continuity make no more than close contact and interlock mechanically. A comparison of false and of true ankylosis is presented in two drawings in *Fig.* 607.

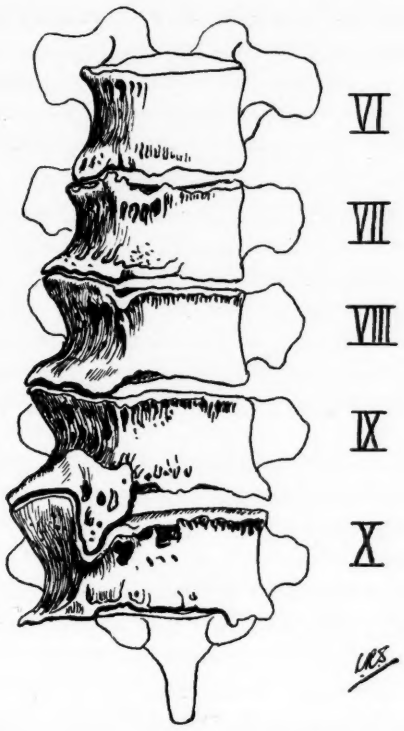


FIG. 606.—The vertebræ from the 6th to the 10th thoracic of the same column as is shown in *Fig.* 597. The drawing is intended to show the ' purposive ' fashion in which osteophytes grow towards each other from adjacent vertebral margins. Between the 9th and 10th vertebræ the osteophytes have developed an interlocking mechanism.

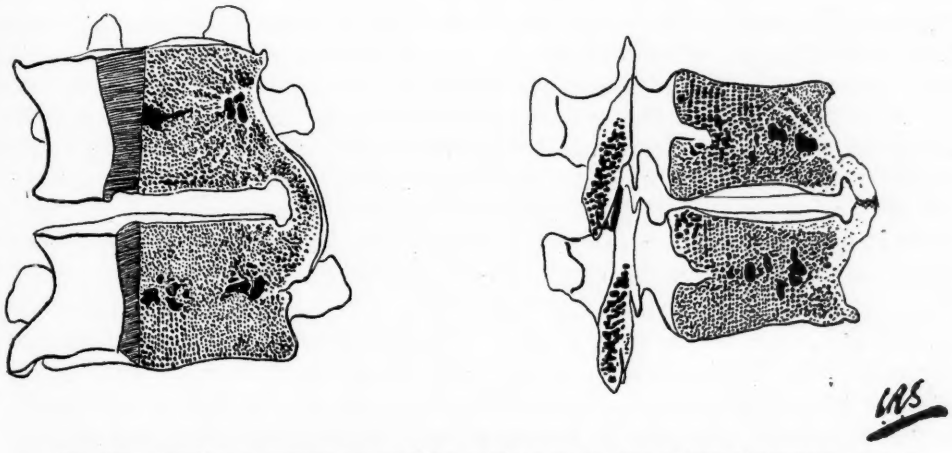


FIG. 607.—Drawings to contrast true and false ankylosis on the basis of internal structure. On the left is shown a section, drawn from a photograph, which passes through the 1st and 2nd lumbar vertebræ of the same column as is shown in *Fig.* 605, after a wedge-shaped piece has been removed. It will be noted that the shell of compact bone is thicker at the pedestals of the bridge and the bridge itself than in the vertebral body. Strands of compact bone traverse the marrow cavity and disorganize the normal structure of the vertebra, but the marrow-containing cavities are continuous from bone to bone. On the right is a section, also drawn from a photograph, which passes through 10th and 11th thoracic vertebræ, whose ankylosis is false. The osteophytes from each of the vertebræ have made contact but not effected bony ankylosis. The osteophytes are composed of compact bone, are based on compact bone which traverses the two vertebræ that bear them in strands, but they do not yet contain marrow cavities. In both these sections note the bulging of the intervertebral space under the osteophytes, and the conspicuous outer epiphysial grooves.

DEGENERATION OF THE INTERVERTEBRAL DISCS

The speculation has been made that degeneration of the nuclei of the intervertebral discs initiates the changes that end in polyspondylitis marginalis osteophytica. Strain in the deep intervertebral ligaments and osteophyte formation in them occur as the discs bulge and the vertebral bodies slide or rotate on each other.

The causes of degeneration of the intervertebral discs demand inquiry.

Arbuthnot Lane,[4] in his essays on labour changes, stated that as a consequence of carrying heavy loads, the vertebræ acquire density and the intervertebral discs are compressed and narrowed. Kaufman[5] has made the same observation more recently. On the evidence of radiograms Ross-Smith states that thinning of the intervertebral discs precedes the appearance at the vertebral margins of ' lipping ', which may be identified with polyspondylitis marginalis osteophytica. It seems, then, that one cause of degeneration of the intervertebral discs may be traumatic, i.e., overweighting.

Ross-Smith also confirmed Beadle's observation that degeneration starts ' naturally ' after the fourth decade of life and in its proportional incidence follows that of arteriosclerosis. A second cause of degeneration of the intervertebral discs, then, may be called ' senile '.

I surmise that a third cause may exist—namely a toxic one. There is need for research in making clear the physical path by which nutritive fluids reach the intervertebral discs. The cancellous marrow spaces of the vertebral bodies lack bony ' end-plates ' and are closed by the cartilage plates with which the intervertebral discs are in contact. It is supposed that through these cartilage plates lymph-like fluid perfuses and bears the means of nutrition to the discs, which have no known blood-supply. If this be so, microbial or toxic invasion of a disc implies previous invasion of the vertebral body by microbes or toxins. It is further implied that the toxin invading the vertebral body is less virulent than that which causes osteoporosis and collapse, or else is selective of disc nucleus tissue. The resistance of the bone is likely to be better than that of the disc because of its blood-supply. The bone has to supply the osteoblastic cells which initiate osteophyte growth in ligament.

I can briefly outline the course of a case of severe ' lumbago '. X-rays showed narrowing of certain intervertebral discs and early growth of osteophytes from vertebral margins. A febrile illness and the known association of this change in the discs with typhoid infection led to a search for that organism. *B. typhosus* was not found, but a hæmolytic *B. coli* was isolated and a vaccine was prepared. The result of administration of the vaccine was striking; the patient's condition improved remarkably and a cure was effected. Subsequent X-ray examination showed the intervertebral discs to have recovered their proportions and the osteophytes to have become static. Such a case suggests that a toxic or a bacterial infection may cause spondylitis marginalis osteophytica secondary to disease of the intervertebral disc. It must be left for those with greater opportunities for clinical and pathological research to confirm or to reject the foregoing speculations.

SUMMARY AND CONCLUSIONS

Polyspondylitis marginalis osteophytica, or, more shortly in English, marginal spondylitis, is the familiar condition in which the vertebral bodies bear osteophytes at their margins or show ' lipping ' in X-ray films.

The osteophytes are shown to arise at a constant position on the vertebral bodies, being separated from the flat surfaces of the vertebral bodies by grooves which mark the outer edges of the epiphyses. These grooves receive the sheaths of the intervertebral discs. The osteophytes themselves arise in the short deep ligaments that connect vertebral body to vertebral body. In consequence the osteophytes lie between the intervertebral discs and the superficial fibrous system which envelopes the whole series of vertebral bodies and includes the anterior and posterior common ligaments.

The distribution in the vertebral column of osteophyte-bearing vertebræ, when set out in graphic fashion, makes a three-waved curve with three districts of maximum incidence and three minimum points. The minimum points are at the 'anticlinal' vertebræ through which a plumb-line would fall in the erect attitude of the body. These vertebræ are supposed to be balanced and with a minimum tendency to slide or to rotate.

It is suggested that ossification is the result of strain put upon the short deep intervertebral ligaments when the nuclei pulposi of the intervertebral discs lose their normal turgid elasticity. Loss of turgescence permits intervertebral discs to bulge and vertebræ to slip or rotate upon their neighbours. Any of these events throw strain upon the deep intervertebral ligaments, and evidence of their occurrence is brought forward. The chain of circumstances that ends in the production of osteophytes is thought to commence in degeneration of the nuclei of the intervertebral discs. This degeneration may be brought about by trauma, i.e., by overweighting, by 'natural' senile change, or, perhaps, by the invasion of toxins.

The skeletons stored in the Anatomy Museum of the University of the Witwatersrand were those on which the first observations were made. I owe grateful thanks to Professor R. A. Dart and to the University of the Witwatersrand for the facilities afforded me. The work was continued on a large collection of vertebral columns in the Anatomy Museum at Cambridge. For opportunities of extended research my thanks are due to Professor J. T. Wilson and to Dr. W. L. H. Duckworth. Dr. A. Sutherland Strachan, Pathologist to the Johannesburg General Hospital, very kindly supplied the material from which I have studied the anatomy of the soft tissues of the vertebral column. Sir Walter Langdon-Brown, Regius Professor of Physic in the University of Cambridge, and the M.D. Committee permit me to publish these observations, which formed part of a thesis submitted for the degree of M.D. during 1933.

REFERENCES

[1] BEADLE, ORMOND A. *The Intervertebral Discs*, Medical Research Council Special Report Series No. 172, 1931.
[2] FISHER, TIMBRELL, *Chronic Non-tuberculous Arthritis*, 1929.
[3] ROSS-SMITH, "The Intervertebral Discs", *Brit. Jour. Surg*, 1931, xviii, 358.
[4] LANE, ARBUTHNOT, "Some Points in the Physiology and Pathology of the Changes produced by Pressure in the Bony Skeleton of the Trunk and Shoulder Girdle", *Guy's Hosp. Rep.*, 1886, xliii.
[5] KAUFMAN, *Pathology*, 1929, ii.

EXPERIMENTAL SURGERY

SYMPATHECTOMY IN THE TREATMENT OF ACHALASIA OF THE CARDIA

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(*Being a Hunterian Lecture delivered at the Royal College of Surgeons on January* 30, 1935)

THE choice of a title for this paper has been somewhat difficult, as it appears that included amongst most series of cases of achalasia of the cardia or cardiospasm are three separate entities having a different pathological basis for their causation: (1) Achalasia of the cardia; (2) Cardiospasm; (3) Hypertrophic stenosis of the cardia. The term 'achalasia' or absence of relaxation is therefore used in its widest sense to cover the common factor in all these three conditions—absence of relaxation of the cardia—and not to denote a definite pathological entity.

The clinical picture of achalasia of the cardia or cardiospasm is sufficiently well-known not to merit any further discussion in this paper. Reference must, however, be made to the nature of the obstruction met with. It is generally accepted that the most common site of the obstruction is at the level of the diaphragm. In a few cases, however, the dilatation may be seen to extend as far as the cardiac orifice of the stomach, as in three of Moore's[1] fourteen cases, and more rarely the obstruction may be well above the diaphragm (*see Fig.* 612). If the obstruction is due to the presence of an intrinsic sphincter, the whole lower inch or two of the œsophagus must partake of this sphincteric action, in order to account for the variable site of the obstruction. The presence of an anatomical sphincter is extremely difficult to demonstrate and has frequently been denied. However, Shattock[2] has shown two preparations which exhibit a widespread thickening of the circular muscle extending on to both œsophagus and stomach and situated entirely below the level of the diaphragm. Poulton and Payne[3] describe a similar thickening in the lower portion of the œsophagus, and remark that it is noteworthy that that part of the œsophagus which partakes in the sphincteric action extends for some little distance above the diaphragm. It is of interest that in these observations the apparent sphincteric area surrounds a length of the lower portion of the œsophagus and is not sharply localized. In post-mortem examinations on typical cases of achalasia of the cardia or cardiospasm it is never possible to demonstrate any muscular hypertrophy in this sphincteric region. Despite the severity of the obstruction met with during life, the condition has been classically defined by Walton[4] as one of "dilatation and hypertrophy of the œsophagus in which at post-mortem examination no obstruction can be found distal to the dilatation." Whatever it is that causes obstruction during life therefore ceases to act after death.

In contrast to this is a type of case described by Moore and Brown and Kelley[5] which simulates achalasia of the cardia in its symptoms and X-ray appearances

(*see Fig.* 616), but differs from this condition in that there is a muscular hypertrophy of the cardiac sphincter similar to that seen in congenital hypertrophic pyloric stenosis. In cases of this type, which are little recognized and are extremely rare, the obstruction does not disappear after death, but persists, so that it is possible to distend the œsophagus with water. The cardiac sphincter in these cases surrounds a narrow canal over an inch in length, the walls of which are greatly thickened. Such a condition can obviously not be due to any inco-ordination of the nervous control of the cardia, and stands in the same relationship to achalasia and cardiospasm as does congenital hypertrophic pyloric stenosis to pylorospasm, which latter conditions have been shown to be entirely separate entities.

If we leave aside this separate group of hypertrophic stenosis of the cardia and confine our attention to the true type as defined by Walton, we are immediately confronted with a variety of theories to account for the obstruction. These may be conveniently considered according to whether they postulate changes in the œsophagus itself or in adjacent structures. In the latter group Mosher[6] believes that deformity of the œsophageal groove on the posterior surface of the liver alone or together with kinking of the œsophagus is responsible for the obstruction. Hill[7] and Jackson[8] regard an inco-ordination of the normal movement of the diaphragm during deglutition as the causative factor, Jackson postulating an actual phrenospasm; but the obstruction is not always at the level of the diaphragm, it may be above it or below. The recent work of Fulde[9] is of interest in relation to this theory. This investigator describes an intrinsic sphincter mechanism in the lower portion of the œsophagus the action of which is normally modified by the phrenico-œsophageal membrane. This structure in virtue of its high attachment to the œsophagus closes the cardia by compression during descent of the diaphragm. To cause an obstruction of the œsophagus the diaphragm would therefore have to be fixed in the inspiratory position. Theories based upon changes occurring primarily in the œsophagus include the view expressed by Shaw and Woo,[10] that kinking of the œsophagus produces the obstruction. This kinking is seen only in the more advanced cases where a combination of dilatation and elongation has deformed the œsophagus, and as this elongation results from the obstruction kinking cannot be the causative factor. As Walton has stated, the early cases are always of the fusiform type in which the cardia is in the most dependent position.

Finally, we come to those who believe that the obstruction is due to inco-ordination of the nervous mechanism of the cardia. Hurst[11] affirms that there is a paralysis of the vagal opening mechanism. Walton states that there is no paralysis but rather a spasm of the part (presumably he considered it as mediated through the sympathetic fibres), for which he suggests a congenital origin. The difference in their views is well illustrated in relation to the passage of bougies, for whereas Hurst maintains that a bougie will always pass the sphincter readily without resistance, Walton states that it is firmly gripped in its passage. For either of these views to be effective there must exist at the cardia a true intrinsic sphincter the tonus of which may be modified by the action of extrinsic nerves. A brief survey of the literature reveals a considerable discrepancy in the results obtained by experimental investigation of both these points. In a recent paper[12] the previous experimental evidence was summarized and the results of an investigation of the innervation of the œsophagus were published in full. The experiments were conducted on cats whose œsophagus as shown by Arey and Tremaine[13] corresponds most closely in structure to the human.

By stimulation experiments it was shown that the interdiaphragmatic and intra-abdominal portions of the œsophagus function as a true intrinsic sphincter which is relaxed by the vagus and contracts on sympathetic stimulation. The sphincter is capable of functioning independently of surrounding structures and receives its sympathetic supply from the cœliac plexus in fibres which follow the course of the left gastric artery and its œsophageal branch to the lower end of the œsophagus.

Excision of the extrinsic nerves supplying the œsophagus, as studied by X-rays taken of bismuth meals, showed that vagal excision reproduced the X-ray pathological and clinical picture of achalasia of the cardia. The sphincter failed to relax despite recovery of some peristalsis. These results were permanent, the changes produced being proportional to the extent of the denervation performed and the length of time elapsing after operation.

If, however, both thoracic sympathetic chains were excised at the same time as the vagi were divided, no obstruction resulted at the cardia, the sphincter showing

FIG. 608.—X-ray showing the result of double vagotomy, seven months after operation. There is marked obstruction at the sphincter

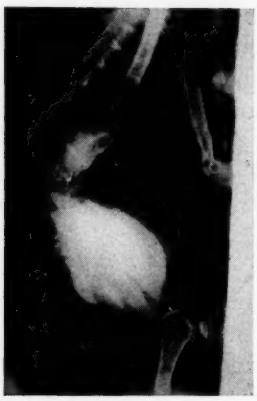

FIG. 609.—X-ray showing relief of the obstruction at the sphincter following cœliac sympathectomy. (Cf. *Fig.* 608.)

at first diminished tonus which later recovered somewhat but always relaxed to allow the meal to enter the stomach. Denervation of the sympathetic fibres which supply the cardiac sphincter by the operation of cœliac sympathectomy resulted in a complete loss of sphincter tonus, so that the meal could be caused to regurgitate from stomach to œsophagus. Later the tonus recovered to some degree but there was abnormally rapid passage of the meal through from œsophagus to stomach.

In animals in which achalasia of the cardia had been produced by a vagal section, subsequent cœliac sympathectomy resulted in a complete relief of symptoms, and X-rays now showed the sphincter relaxing and the meal entering the stomach without obstruction (*Figs.* 608, 609).

Animals treated by these means have now been followed for one year after sympathetic excision. There has been no onset of obstruction in animals with combined vagal division and thoracic sympathetic excision. In animals in which sympathectomy was performed after the development of achalasia there has been no recurrence of symptoms and a progressive gain in weight. X-rays still show the meal passing readily through the sphincter, with rather marked increase in the

peristalsis of the lower third of the œsophagus. The success of sympathectomy is to be judged on this relaxation of the sphincter rather than on regurgitation from the stomach to the œsophagus, which disappears as the muscle recovers tonus.

The bearing of these results upon the production of achalasia or cardiospasm is interesting. Bilateral vagal section if complete produces the symptoms and X-ray and pathological appearances of achalasia of the cardia. But if, however, the sympathetic fibres passing to the œsophagus are removed at the same time as the vagal section is performed, no obstruction results, and following the production of the obstruction it may be relieved by sympathectomy.

It follows, therefore, that whether we subscribe to the views of Hurst, that the obstruction is due to vagus failure, or those of Walton, that it is due to a spasm, in both cases the integrity of the sympathetic supply is necessary for the obstruction to occur, and therefore it should be relieved by sympathectomy.

CLINICAL PATHOLOGY

The study of 66 cases in various hospitals has shown that amongst them are some in which a bougie passes the sphincter readily despite severe clinical and X-ray evidences of obstruction, presumably an achalasia or vagus failure. In others of the spasmodic type the bougie is firmly gripped in passing, and in these the symptoms sometimes show a characteristic intermission. It is therefore felt

FIG. 610.—Section of lower end of œsophagus from a case of achalasia or cardiospasm. There is superficial œsophagitis. The region of Auerbach's plexus is free from inflammatory changes.

that all cases of achalasia have not the same pathogenic factor and that it is desirable to separate them into three groups: (1) Cases of vagus failure—achalasia; (2) Cases of spasmodic contraction (cardiospasm); (3) Hypertrophic stenosis of cardia. The latter is not truly an example of achalasia for the reasons already stated, but, being extremely liable to be mistaken for it, it is included here for the sake of further emphasis.

Under the first group, lesions of the vagus trunk are rare. Kraus[14] has described a case in which the vagus nerve was degenerated. In another Politzer[15] described involvement of the vagus by a mass of glands. Although isolated cases of trunk lesions are described, the main site of vagus involvement is at the ganglia of Auerbach's plexus, in which Rake[16] has demonstrated chronic inflammatory changes. These show a progression from round-celled infiltration to degeneration of ganglion cells and ultimately to complete fibrosis. The importance of these changes is, that these ganglion cells constitute vagal relays, a view held by such authorities as Catherine Hill,[17] Kuntz,[18] and Abel.[19] Dr. Rake has allowed me the privilege of examining his preparations, and the changes shown in these are most striking.

Cases, however, occur which do not show degenerative changes. An example of this type is shown in *Fig.* 610, obtained from a woman who died during a thyroid crisis but had incidentally achalasia. In this case there is a superficial œsophagitis, but the region of Auerbach's plexus is not affected. This merely shows that all cases have not a common cause, without in any way disproving a very definite factor in the production of the disease. The changes in Auerbach's plexus, as pointed out by Gask and Ross,[20] may have a definite bearing upon the treatment of this condition by sympathectomy. "Should it be established, as is commonly supposed, that an inflammatory or degenerative lesion of Auerbach's plexus is invariably present, division of the sympathetic supply proximal to Auerbach's plexus would not be expected to be beneficial." Alternatively, if the normal function of Auerbach's plexus is interfered with, sympathectomy, while producing diminished sphincter tonus, might still leave the tube dilated.

In considering cases of spasmodic type Walton, in his Jacksonian Essay on cardiospasm, discards cases of hysterical spasm in young women, as these fail to show the dilatation so characteristic of the disease. Similarly cases of local reflex spasm from such causes as œsophagitis, peptic ulcer, and following upon such operations as gastro-enterostomy, although of interest, are usually transient or disappear on the cure of the causative lesion.

True examples do, however, recur. One case to our knowledge succeeded upon a perineal excision of the rectum and presented marked symptoms. In another (*see Fig.* 612) there was a recurring and extremely severe spasm attended by dilatation of the œsophagus in a young man following upon a perforated duodenal ulcer, and in this patient the bougie could be felt to be firmly gripped in its passage. Walton goes further and considers that the majority of cases are due to spasm.

TREATMENT

The various forms of treatment at present used are summarized here, and are sufficiently familiar to need but little amplification.

1. Dilatation of the cardia
   - Hurst's mercury bougie
   - Œsophagoscopy and bougie
   - Hydrostatic dilators
   - Digital dilatation
2. Plastic operations on the œsophagus
   - Reisinger
   - Freeman
3. Plastic operations on the cardia
4. Short-circuit operations

For dilatation of the cardia Hurst advocates the passage of a rubber bougie of large diameter weighted with mercury which he says in all cases passes the sphincter readily, and at one time stated that the condition could always be relieved without operation. Alternatively a solid bougie may be passed from the mouth under direct vision by œsophagoscopy. The passage of such an instrument blindly cannot be too strongly condemned. Various forms of hydrostatic dilators, such as those of Gottstein, Plummer, and Mosher, have been devised. These consist essentially of a hollow tube with an inflatable bag surrounding its terminal portion. This may be introduced into the cardia and distended. The degree of stretching is controlled by the incorporation of a manometer in the system if water is used. Alternatively the system may be air-containing, and the sense of resistance used as an indication of pressure exerted. A more extensive dilatation is secured by the operation of gastrostomy and digital dilatation in which the cardia may be sufficiently stretched to accommodate three fingers. Plastic operations on the œsophagus are of historic interest. They have been designed to correct either the dilatation (Reisinger) or the lengthening of the œsophagus (Freeman). Plastic operations on the cardia are of two types. In the first, which is similar to the Heineke-Mikulicz pyloroplasty, a longitudinal incision is made and closed transversely. Heller's incision is similar to Rammstedt's operation for congenital hypertrophic pyloric stenosis. But since in typical cases there is no muscular hypertrophy of the cardia this procedure appears to be ill-founded. Short-circuit operations consist essentially in the formation of an œsophago-gastrostomy either above or below the diaphragm.

In order to arrive at an assessment of the value of these methods in general hospital practice an inquiry was undertaken. Sixty-six cases were traced and their present condition ascertained. As some patients had more than one form of treatment 85 results are available for comparison. It is not suggested that these are at all typical of the results to be obtained by dilatation in cases of a mild degree. All the records were obtained from surgical wards, and therefore presumably include the more severe variety.

Type	No.	Cure	Improvement	Mortality
Mercury bougie	28	2	8 cases	–
Œsophagoscopy and bougie ..	16	–	Up to 1 year	3 (20 per cent)
Dilators	5	–	7 months to 1 year	–
Digital dilatation	26	8	2 months to 3 years	3 (11 per cent)
Heller's incision	2	–	1 month to 3 months	–
Cardioplasty	1	–	1 year	–
Gastrostomy	5	–	—	–
Œsophago-gastrostomy	3	3	—	–

Treatment by Hurst's bougie gave a complete cure in 2 cases; 8 were much improved, with gain in weight. In the remainder the relief was incomplete, the symptoms recurring if the bougie were discarded, the tube being used as often as two to three times a day. Those patients who did not find passing the tube impossible complained of the inconvenience or depression caused by its repeated passage, and in some cases of the attacks of œsophagitis which this produced.

Dilatation by bougies resulted in three deaths, two from empyema and one from perforation of the stomach. As this occurred even under direct vision it is a strong point against the blind passage of an œsophageal bougie. Gastrostomy and digital dilatation was completely successful in eight cases up to the time of inquiry (3 to 7 years). The remainder showed recurrence of symptoms in periods varying from two months to three years after operation. Although the recurrent symptoms were less severe in some cases, they nevertheless required further treatment by a bougie. The fact that gastrostomy alone was performed in 5 cases shows that contrary to Hurst's statement it is definitely impossible to pass the sphincter in certain cases. Three cases of œsophago-gastrostomy all showed complete relief on a slightly restricted diet after three years. It appears from these figures that the various forms of treatment by dilatation are not completely successful. The period of relief appears to be proportional to the initial degree of stretching, being longest in the case of digital dilatation. Plastic operations have little to recommend them; the most uniformly successful operation is œsophago-gastrostomy, which is a procedure of some magnitude.

In the treatment of this condition by sympathectomy, Recalde[21] described an operation designed to remove Auerbach's plexus, which he regards as responsible for maintaining the obstruction. To effect this, the external muscular coat was stripped from the sphincteric area and the underlying plexus destroyed. Such a procedure must also interrupt the sympathetic supply to the circular muscle of the cardia. Of the 4 cases in his series, 1 died from perforation, but 3 remained completely relieved of their obstruction. Although these results encourage the possibility of sympathectomy, the method has none of the advantages of a remote neurectomy. From the experimental findings, it has been shown that the sympathetic supply to the cardia may be approached either by excision of the lateral thoracic sympathetic chains or by interruption of the fibres along the course of the left gastric artery. Kuntz[22] states that in man the œsophagus is innervated chiefly from the inferior cervical ganglia, further branches also passing to the œsophagus from the thoracic sympathetic ganglia. Hovelacque[23] considers that these thoracic branches are few in number and not to be found in all subjects. Where they occur they are more numerous above the origin of the great splanchnics. It is probable that certain of these fibres may in part contribute to the sphincteric supply through the periœsophageal plexus. Their denervation could be effected by a bilateral cervico-thoracic ganglionectomy designed to include the upper thoracic ganglia. Since such a procedure interrupts the sympathetic supply to the head, upper limbs, and heart, with the production of a bilateral Horner's syndrome, it appears to be a mutilating procedure. The report of one case treated by this means has been made recently from the Mayo Clinic.[24] The patient obtained symptomatic relief, but a bilateral Horner's syndrome was produced. An X-ray of the condition of the œsophagus following operation is not published. Denervation of the left gastric artery appears to be preferable provided that a sufficient denervation is obtained. Evidence that this operation affects the human cardia as in cats was obtained from a case of cœliac sympathectomy, the first on record, performed by Mr. J. B. Hume,[25] of St. Bartholomew's Hospital, in the treatment of gastric ulcer. Two years after operation this patient was recalled and X-rayed. There was seen to be abnormally rapid passage of the meal through the cardia.

Dissection in the human shows that in order to interrupt the fibres passing

to the cardia, it is not necessary to strip the trunk of the cœliac axis artery. The cardiac sphincter supply is derived chiefly from the left side of the cœliac plexus. Fibres from the right side are distributed mainly along the hepatic branch, a few pass along the left gastric artery.

From the cœliac plexus fibres pass in a broad leash towards the left gastric artery, joining at various levels along its course and forming a network in the surrounding fat, the majority lying posterior and to the right of the vessel. From this network two main divisions form, which are distributed with the left gastric artery to the stomach and with the œsophageal branch to the cardia. This distribution is shown in *Fig.* 611, a drawing made of the portion of tissue removed in the course of operation. To effect a denervation it is necessary to excise the left gastric artery with its surrounding fat and nervous tissue. Division of the artery alone

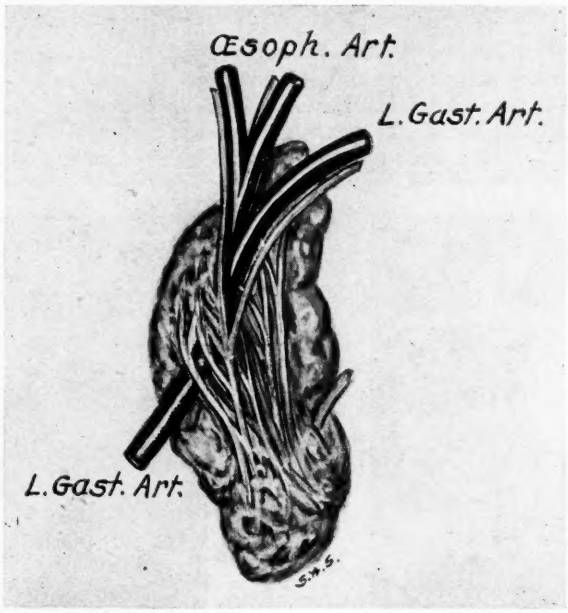

FIG. 611.—Drawing of portion of tissue excised at operation. This shows the large number of fibres passing to join the left gastric artery, forming a plexus on its anterior and posterior surface. The majority of the fibres pass posteriorly.

is obviously inadequate. Division of the trunks alone fails to interrupt the peri-arterial network. Rarely a branch may be seen as described by Brandt,[26] which passes directly from the left cœliac ganglion to the cardia and not along the course of the vessels.

The approach to these structures is made through the lesser omentum, and in all cases it is necessary to excise the left gastric vein in order to obtain an exposure. The incision may be a left paramedian, or, as illustrated in *Fig.* 616, a double subcostal incision may be employed. The latter incision gives an excellent exposure.

During August, 1934, a suitable case for operation was found in the wards of the Royal Infirmary, Edinburgh. Mr. W. A. D. Adamson[27] carried out the operation. Since that time he has operated on three cases. For his readiness to

apply this new operation and his generous collaboration in keeping me informed of his results, I am deeply indebted.

It was not until November, 1934, that the first suitable case became available in the Surgical Unit at St. Bartholomew's Hospital, London. Professor G. E. Gask undertook the operation, and has since operated on three additional cases. I am deeply grateful for his continued advice and suggestions during the course of this work.

The cases described are not intended to form a complete series. Three examples (one of each of the three types described on p. 867) are chosen to form a preliminary report.

CASE REPORTS

Case 1.—Male, aged 27. In November, 1933, suffered from a perforation of a duodenal ulcer. Convalescence complicated by rupture of the abdominal wound. In May, 1934, he returned complaining of persistent vomiting. He was emaciated and had acetonuria. X-rays showed spasmodic contraction of the lower œsophagus with obstruction above the level of the diaphragm. He was treated on three occasions by œsophagoscopy and the passage of bougies, which were felt to be firmly gripped at the sphincter. The relief

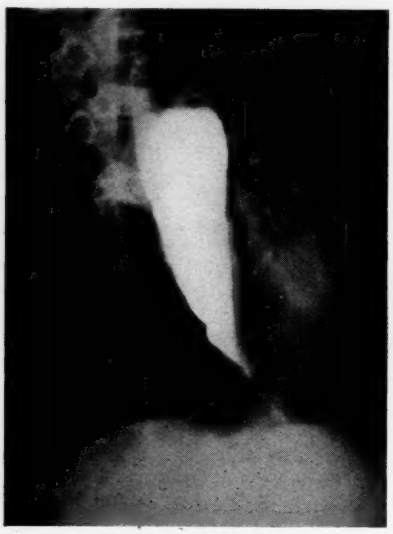

FIG. 612.—*Case* 1. (Mr. Adamson's.) X-ray before operation (Aug. 12, 1934), twenty minutes after swallowing. Shows contraction of the lower œsophagus extending above the level of the diaphragm. There is dilatation of the œsophagus with typical cone-shaped termination at its lower end.

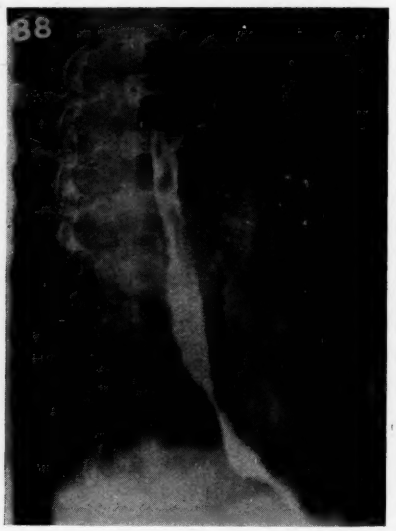

FIG. 613.—*Case* 1. After operation (Dec. 12, 1934), eight seconds after swallowing. Shows the meal passing readily through the site of previous obstruction. The dilatation has subsided.

afforded by this means showed a progressive decrease on each occasion from three weeks to one week, and on the last occasion to four days. On August 4 operation was performed, his weight then being 9 st. 2 lb. Since operation he has remained perfectly well and can eat anything. His present weight is 10 st. 8 lb. *Fig.* 612 shows the œsophagus before operation, twenty minutes after swallowing. *Fig.* 613 shows the œsophagus five months after operation, eight seconds after swallowing. The meal can be seen passing readily through the previously obstructed segment.

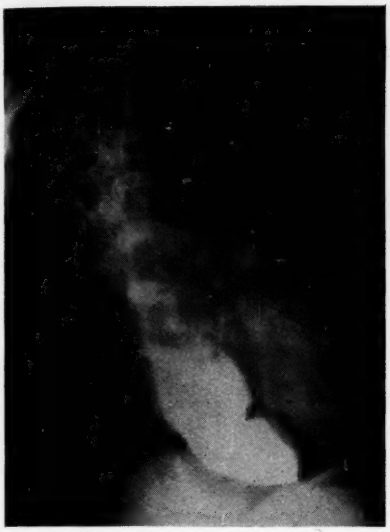

FIG. 614.—*Case* 2. (Mr. Adamson's.) X-ray before operation (Nov. 8, 1934), twenty minutes after swallowing. Shows obstruction at the level of the diaphragm, with dilatation of the œsophagus.

FIG. 615.—*Case* 2. After operation (Dec. 20, 1934), eight seconds after swallowing. Shows diminished sphincter tonus with the meal passing into the stomach. There is still some dilatation.

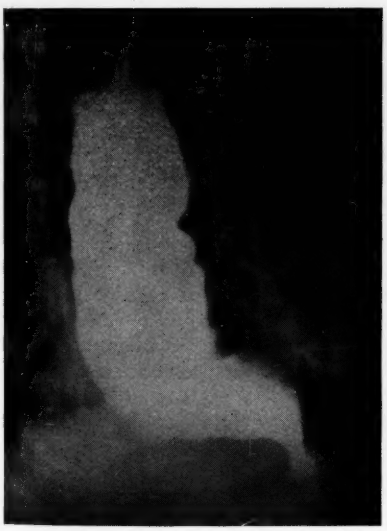

FIG. 616.—*Case* 3. (Professor G. E. Gask's.) X-ray before operation (Nov. 14, 1934), ten minutes after swallowing after the previous removal of 8 oz. of residual fluid. Shows dilatation of the œsophagus with obstruction below the level of the diaphragm.

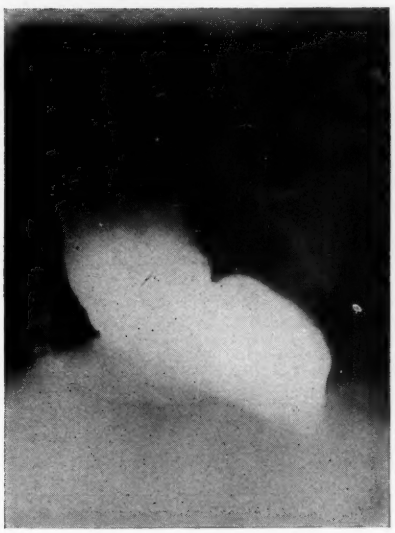

FIG. 617.—*Case* 3. X-ray six weeks after operation, ten minutes after swallowing. The dilatation and residual fluid were diminished, but obstruction still persists at the cardia.

Case 2.—Male, aged 28. April, 1934. Following upon pneumonia he developed symptoms of vomiting and dysphagia. X-rays on Nov. 8 showed the œsophagus to be dilated, with obstruction at the level of the diaphragm. As a diagnostic procedure bilateral injection

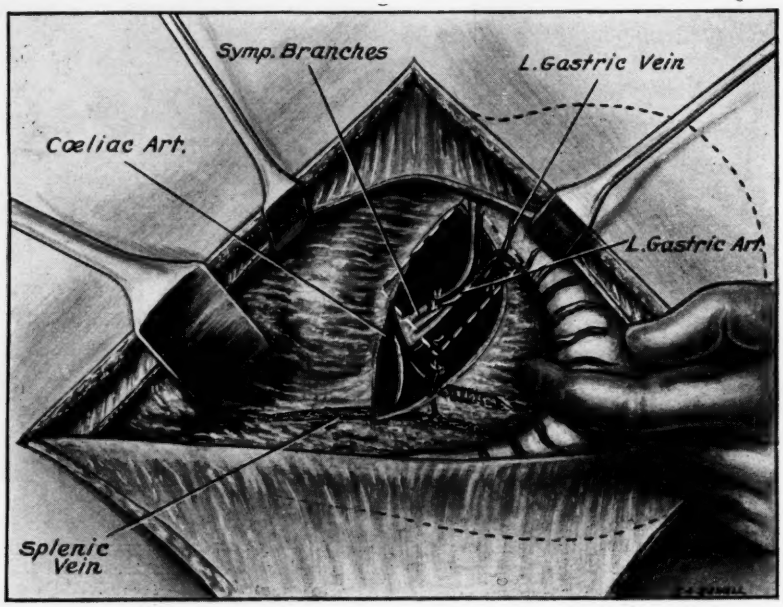

FIG. 618.—*Case* 3. Operation: double subcostal incision. The left lobe of the liver has been mobilized and retracted to the right. The lesser omentum has been incised and the left gastric vein divided. The left gastric artery is divided at its origin. The portion of tissue to be excised is included in the white frame.

of the stellate ganglia was performed and was seen to have no effect upon the outline or peristalsis of the œsophagus. Operation was performed on Nov. 12. Since operation swallowing has improved and he is gaining weight. *Fig.* 614 shows the condition before operation, twenty minutes after swallowing. *Fig.* 615 shows the condition after operation, eight seconds after swallowing. There is diminished sphincter tonus and the meal is passing into the stomach.

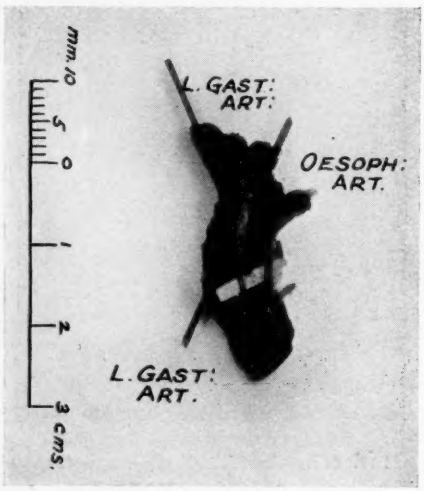

FIG. 619.—*Case* 3. Photograph of a dissection of the tissue excised at operation: posterior aspect. There has been some shrinkage owing to fixation.

Case 3.—Female, aged 47. Seven years' history of dysphagia and vomiting. Treated for five and a half years by the regular passage of bougies without permanent relief. Loss of weight from 16 stone to 11 st. 10 lb. X-ray (*Fig.* 616) after the removal of 8 oz. of residual fluid from the œsophagus showed marked dilatation, with obstruction below the level of the diaphragm. The meal was held for half an hour with the passage of only a minute quantity to the stomach. Operation on Nov. 20, 1934. A double subcostal incision was employed (*Fig.* 618) and the left gastric vein divided. The left gastric artery was excised from its origin to the termination of the œsophageal and gastric branches, together with surrounding fat and nervous tissue (*Figs.* 619, 620). Palpation of the cardiac region showed that the sphincter was enormously thickened,

approximately the size of a man's thumb, and over an inch in length. During this palpation the thickened area was felt to relax twice in the course of one minute at the close of the operation. After operation swallowing was improved, the patient taking a full diet regularly without regurgitation. There was, however, an occasional sense of obstruction, but this was less marked. X-rays six weeks after operation showed diminished dilatation of the œsophagus, but there is still obstruction at the cardia (*Fig.* 617). The sense of obstruction is now increasing.

FIG. 620.—*Case* 3. Section of the tissue excised, showing a large number of non-myelinated trunks surrounding an artery. It will be noticed that there is a concentration of the nerve trunks in one area, actually posterior and to the right of the vessel.

The first case is of spasmodic type and shows complete relief after neurectomy. The second case appears to be a true achalasia; bilateral injection of the stellate

ganglia did not affect the peristalsis of the œsophagus. This case shows diminished sphincter tonus, but there is still some dilatation. The third case is an example of hypertrophic stenosis of the cardia. As the obstruction in this condition persists after death it could not be completely relieved by neurectomy.

SUMMARY

1. Included amongst most series of achalasia of the cardia or cardiospasm are three conditions, true achalasia, cardiospasm, and hypertrophic stenosis of the cardia.

2. The first two result from autonomic imbalance in the control of the cardia and are improved by sympathectomy. The third stands in the same relationship to achalasia or cardiospasm as does hypertrophic pyloric stenosis to pylorospasm. It is a separate entity and shows little improvement following sympathectomy.

3. The operation of left gastric sympathectomy affords relief in cases of the first two types.

4. Details of the operation are given, and the results of the operation in cases of each type are described.

I wish to express my thanks for permission to use cases from Professor Gask's and Mr. Adamson's series.

REFERENCES

[1] MOORE, *Proc. Roy. Soc. Med.* (Sect. of Laryngol.), 1918–19, xii, 68.
[2] SHATTOCK, *Ibid.*, 92.
[3] POULTON and PAYNE, *Quart. Jour. Med.*, 1923–4, xvii, 60.
[4] WALTON, *Brit. Jour. Surg.*, 1924–5, xii, 701.
[5] BROWN and KELLEY, *Proc. Roy. Soc. Med.* (Sect. of Laryngol.), 1918–19, xii, 48.
[6] MOSHER, *Laryngoscope*, 1922, xxxiii, 348.
[7] HILL, *Proc. Roy. Soc. Med.* (Sect. of Laryngol.), 1918–9, xii, 37.
[8] JACKSON, *Laryngoscope*, 1922, xxxii, 139.
[9] FULDE, *Deut. Zeits. f. Chir.*, 1924, ccxlii, 580.
[10] SHAW and WOO, *Proc. Roy. Soc. Med.* (Sect. of Med.), 1916, Dec., 1.
[11] HURST, *Quart. Jour. Med.*, 1930, xxiii, 491.
[12] KNIGHT, *Brit. Jour. Surg.*, 1934, xxii, 155.
[13] AREY and TREMAINE, *Anat. Record*, 1933, lvi, 315.
[14] KRAUS, *Nothnagel's Spec. Path. u. Therap.*, 1902, 129.
[15] POLITZER, *Münch. med. Woch.*, 1913, lx, 108.
[16] RAKE, *Guy's Hosp. Rep.*, 1927, lxxvii, 141.
[17] HILL, C., *Trans. Roy. Soc.* (Sect. B), 1927, ccxv, 355.
[18] KUNTZ, *Anat. Record*, 1922, xxiv, 193.
[19] ABEL, *Jour. Anat. and Physiol.*, 1913, xlvii, 35.
[20] GASK and ROSS, *Surgery of the Sympathetic Nervous System*, 124.
[21] RECALDE, *Arch. ital. di Chir.*, 1932, xxxii, 613.
[22] KUNTZ, *Autonomic Nervous System*, 1929, 1st ed., 188.
[23] HOVELACQUE, *Anatomie des Nerfs*. 1927, 1st ed., 720.
[24] CRAIG, MOERSCH, and VINSON, *Proc. Mayo Clinic.*, 1934, Vol. ix, 749.
[25] HUME, *St. Bart's Hosp. Rep.*, 1933, lxvi, 28.
[26] BRANDT, *Zeits. f. angewandte Anatomie und Konstitutionslehre*, 1919-20, 302.
[27] ADAMSON, *Proc. Roy. Soc. Med.* (Sect. of Surg.), 1935, January.

SHORT NOTES OF RARE OR OBSCURE CASES

BLACK HAIRY TONGUE OR HYPERKERATOSIS LINGUAE

By Surgeon Captain H. E. R. STEPHENS, O.B.E., R.N.

On Sept. 11, 1934, a Regulating Petty Officer belonging to H.M.S. *Dolphin* was admitted to the Royal Naval Hospital, Haslar. A week previously he had noticed a tuft of black hair on the back of his tongue. This was discovered quite by chance and did not cause any inconvenience. On examination there appeared to

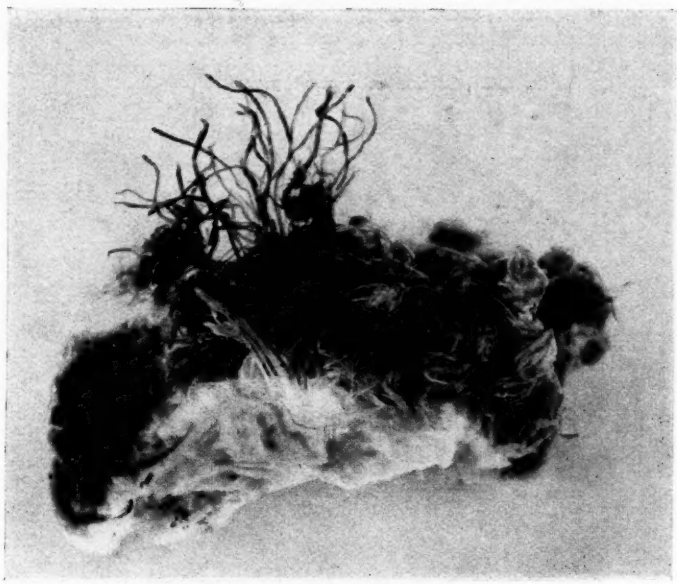

Fig. 621.—Showing condition as excised. (× 2.)

be a ridge of hair on the dorsum of the tongue commencing just in front of the foramen cæcum and circumvallate papillæ; this extended forwards for $1\frac{1}{2}$ in., being about $\frac{3}{8}$ in. wide at its broadest part. On Sept. 14 the growth was excised by Surgeon Commander E. Heffernan. The patient was discharged to duty on Oct. 5, completely cured.

The accompanying microphotographs (*Figs.* 621–623) were taken from the excised specimen by Surgeon Commander J. A. O'Flynn. These show the condition better than any written description. The pathology appears to be obscure; some authorities believe that an infective process is the causal factor, while others

consider it to be a simple hypertrophy with pigmentation of the lingual papillæ. The best account of this interesting disease is given by Andrews[1] and is quoted in full.

" The affection is rare, usually occurring on the dorsum anterior to the circumvallate papillæ, where there are formed black, bluish-black, or brown patches

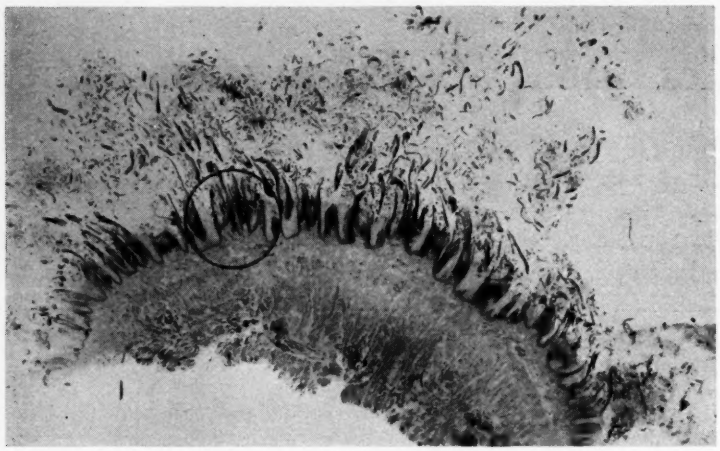

FIG. 622.—Section of excised growth. (× 4.)

FIG. 623.—Section shown in circle in *Fig.* 622 stained by magenta red and picro-indigo-carmine. (× 30.)

consisting of hairlike intertwining filaments from $\frac{1}{4}$ to $\frac{1}{2}$ in. in length. Histologically, Heidingsfeld found elongated and stratified filaments originating from abnormal papillæ within the epithelial covering of the mucous membrane. The cells making up the hairlike processes resemble those on the stratum corneum of

the skin. The colour is due to changes in the horny cells analogous to those which are seen in certain types of hyperkeratosis of the cutaneous surface, or to pigment from substances taken into the mouth. The condition is not due to micro-organisms, but to some congenital abnormality which develops in later life.

" Pseudoblack tongue may originate from local irritants or mouth washes containing coloring matter. Oppenheim demonstrated that hypertrophy and hyperkeratosis of the filiform papillæ in this region can be experimentally produced by daily applications of tinctures containing vegetable coloring matter. He compares the artificially produced condition of the tongue with hyperkeratosis of the skin from tar and aniline products. Prinz believes that the pigmentation of the elongated filiform papillæ is always due to a deposit of pigment from external sources. He was able to demonstrate iron pigment on the filaments of black tongue by chemical tests, and believes the condition is due to a chemical reaction between the hæmoglobin in the blood and certain sulphur and ammonia compounds derived from the decomposition of protein débris or tobacco.

" Another form of pseudoblack tongue is caused by the filamentous mycelia of fungi which grow as molds on the dorsal surface. The diagnosis is confirmed by microscopical demonstration of the fungus and by the ease with which the filaments can be removed from the tongue. Thompson and Montgomery, in a study of two cases, isolated pure cultures of actinomycosis from each. Weidman recovered a similar organism (*Microsporon minutissimum*) in a case of black tongue occurring in a diabetic who also had erythrasma."

REFERENCE

[1] ANDREWS, G. C., *Diseases of the Skin*, 1930, 992. Philadelphia and London.

A RARE TYPE OF VALVULAR OBSTRUCTION OF THE NECK OF THE BLADDER

RECORD OF TWO CASES

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THE presence of valvular folds at the neck of the bladder is an uncommon cause of urinary obstruction, although a number of cases are on record in which valves partially blocked the posterior urethra. In cases of this type a diagnosis is rarely made until prolonged back-pressure and infection have produced irreparable damage to renal function. Of the recorded cases in which the obstructing tissue was wholly intravesical, in that of Harris[1] the valve arose from the posterior margin of the vesical orifice ; in that of Campbell[2] a large fold, termed by him the " trigonal curtain ", arose midway between the vesical outlet and the inter-ureteric ridge ; while in Wallace's[3] case a fibrous partition or diaphragm arose from the

inter-ureteric ridge. In the two cases we are to describe the obstructing fold was attached to the anterior margin of the vesical orifice, an arrangement which, so far as we are aware, has not hitherto been recorded.

Case 1 (J. R. L.).—A man aged 40 years was admitted to the Aberdeen Royal Infirmary on Sept. 2, 1933. He stated that for fifteen years he had had difficulty in passing urine. This was most marked at the beginning of the act; once started, the stream passed fairly easily. On two occasions during this period he had been catheterized to relieve complete retention. Occasionally there had been a scalding sensation during the act of micturition. In other respects he had always considered himself to be healthy.

On Examination.—The patient was a healthy and well-developed man. The tongue was clean, the throat not inflamed. There was no evidence of disease of the nervous system, of the lungs, or of the cardiovascular system. The bladder was distended to an inch below the umbilicus; otherwise abdominal examination was negative. On rectal examination the prostate was small and soft, and the tone of the anal sphincter good. The urine was alkaline and contained albumin; there was a copious deposit of pus. X-ray examination did not disclose any calculus or any osseous deformity of the spine.

On Admission.—The value for blood-urea was 265 mgrm. per 100 c.c. The Wassermann reaction in the blood was negative. An ordinary rubber catheter could be passed with ease, so that the 'admission' diagnosis of stricture of the urethra was obviously incorrect.

Fig. 624.—*Case* 1. The bladder has been laid open, and shows the 'valve' over the anterior margin of the vesical outlet.

For a week he was regularly catheterized three times a day, from 300 to 400 c.c. of urine being withdrawn at each session; in the intervals he passed urine in amounts of 120 to 240 c.c., so that the total daily output was about 2000 c.c. Thereafter, an indwelling rubber catheter was employed, and infusions of 10 per cent glucose solution were given by the intravenous route. Five days after the introduction of the catheter, on Sept. 15, the patient complained of sore throat, and the temperature rose. This condition appeared to be subsiding, and by Sept. 20 the value for blood-urea was 157 mgrm. On Sept. 25, however, the temperature rose sharply, and a patch of erysipelas appeared about the left ear. This spread rapidly; on the morning of Sept. 26 a blood-culture showed a growth of *Streptococcus hæmolyticus*, and the value for blood-urea was 265 mgrm. On Sept. 27 he died.

Autopsy.—Only a partial examination was made. The left kidney itself was small; its capsule was thickened and adherent to the surrounding fat. On section it presented numerous early abscesses and marked tubular degeneration. The left ureter was considerably dilated. Similar changes affected the right kidney and ureter. The bladder was greatly dilated and hypertrophied, although there was little sign of inflammatory change in the mucosa. The ureteric orifices were normal.

A fine membranous band crossed the internal orifice of the urethra from side to side. Anteriorly its base was attached to the anterior margin of the orifice; its posterior margin was free (*Fig.* 624). Microscopically the fold consisted of a mass of œdematous fibrous tissue covered by transitional epithelium.

Case 2 (K. H. W.).—A lad aged 18 years was admitted to the Manchester Royal Infirmary on April 2, 1934. He stated that urination had always been more difficult and more frequent for him than for other boys. Up to the age of 12 years he had had nocturnal incontinence. For five or six weeks before admission his symptoms had been unusually severe; he had to strain very hard to pass urine, and there was marked frequency—every hour or so during both day and night. There was some urgency, but never actual incontinence. Recently he had noticed a tumour in the lower abdomen when the bladder was full, but this disappeared after he had passed urine. Rectal function was not impaired.

On Examination.—The patient was a slender unhealthy-looking youth. A soft swelling, which disappeared on urination, occupied the hypogastrium and part of the right iliac fossa. The external genitalia were normal, and rectal examination showed that the tone of the anal sphincter was normal. There was no evidence of disease of the nervous system. The urine contained innumerable leucocytes, *Bacillus coli*, and a hæmolytic streptococcus. X-ray examination of the urinary tract did not reveal any abnormal shadows.* On admission the value for blood-urea was 98 mgrm. per 100 c.c. He was observed to strain very hard during an act of urination, and passed 240 c.c. of urine with twelve interruptions, which occurred when he relaxed his efforts. Each spurt of urine was of fair force, and appeared slightly more forcible than in cases of complete detrusor paralysis. A No. 15 F. rubber catheter was passed, and 500 c.c. of urine withdrawn, without completely emptying the bladder; it would appear that some of this urine must have come from the upper urinary tract, because the bladder was not palpable before catheterization.

Cystogram (*Fig.* 625).—It was surprising that only a small amount of fluid would run by gravity into the bladder. The main cavity of the bladder appeared to be very small, and round it there were numerous diverticula. There was no evidence of relaxation of the vesical orifice, of the so-called 'funnel urethra', or of ureteral reflux.

Bladder-pressure Estimation (*Fig.* 626).—For twenty-four hours the indwelling catheter, which had been inserted on April 13, was removed. During this period the patient passed some urine, and the swelling in the right iliac fossa again became palpable. The pressures were first recorded during emptying of the bladder, and again during the subsequent filling with fluid. It is probable that the curve of emptying pressures was affected by the overflow of urine from the upper urinary tract. The striking feature was the very steep curve of filling, which was quite abnormal, and previously considered (K. H. W.[4]) to be characteristic of a lesion affecting the sacral segments of the spinal cord or the cauda equina. Evidence of powerful detrusor contraction was lacking, and the pressures achieved during the attempts to urinate (shown by the two lines rising vertically from the curve of emptying) appeared to be due to contraction of the abdominal muscles. A

* A spina bifida was present, but in consultation with Mr. Geoffrey Jefferson it was decided that it had no significance.

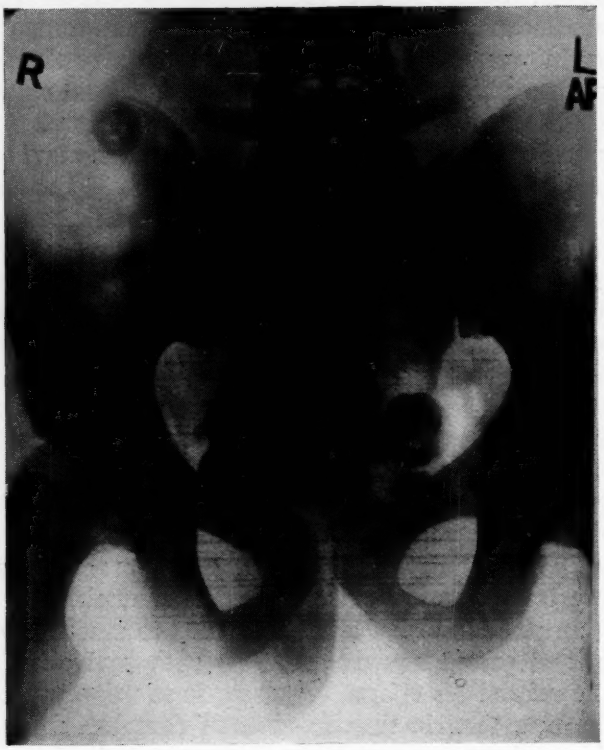

FIG. 625.—*Case* 2. Cystogram.

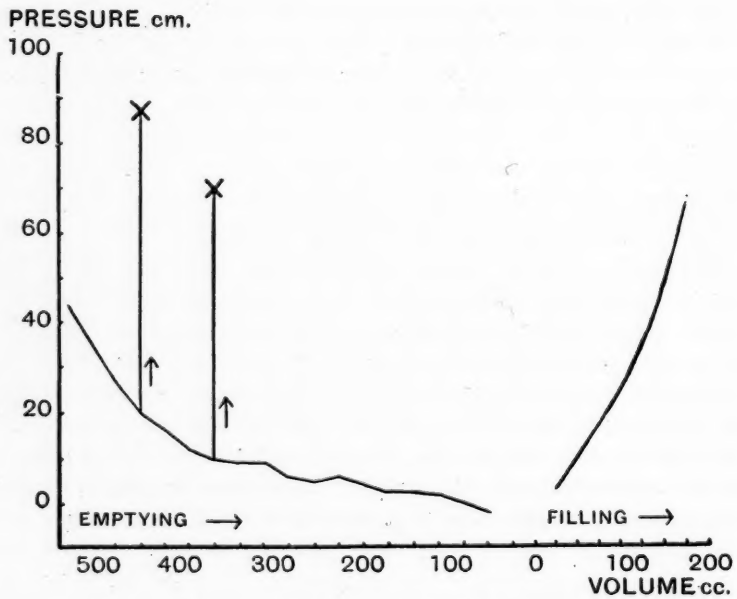

FIG. 626.—*Case* 2. Curve showing emptying and filling pressures in bladder.

definite sensation of filling was noticed after the introduction of 50 c.c. of fluid into the bladder.

Cystoscopy was carried out under ether anæsthesia. The capacity of the bladder was small. Between huge trabeculæ appeared the orifices of numerous diverticula. The right ureteral orifice appeared to be normal, the left was not seen. The trigone was not unduly prominent. Posteriorly the vesical orifice seemed to be normal in outline, but anteriorly and laterally the appearance resembled that of intravesical enlargement of the lateral lobes of the prostate, except that there was no anterior cleft. The posterior urethra could not be seen with the concave sheath. With the convex sheath the posterior urethra appeared to be normal, apart from a slight enlargement of the verumontanum. The prominence which was seen on the anterior aspect of the neck of the bladder did not encroach upon the posterior urethra.

OPERATION.—In order to provide suprapubic drainage, on May 5 suprapubic cystostomy was performed under ether anæsthesia. When the interior of the

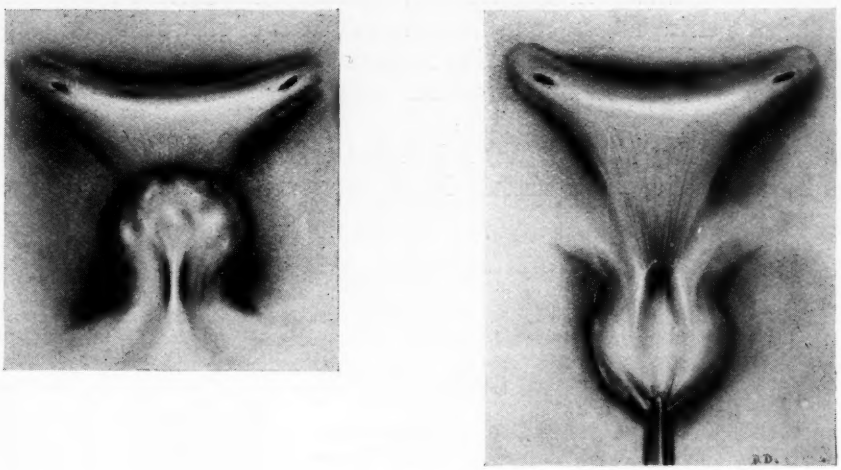

FIG. 627.—*Case* 2. Appearance of valve at operation.

bladder was inspected urine was seen to be issuing freely from both ureters. At the base of the bladder was seen a fold of mucous membrane, 1·5 to 2 cm. high and of similar breadth (*Fig.* 627). It was 6 to 7 mm. in its greatest thickness and soft in consistence. The internal meatus could not be located until a bougie was passed; then it was seen to lie immediately beneath the valvular fold, which appeared to lie naturally over it. The fold was easily and quickly excised, and the bladder was drained by a tube.

For the first three days after operation vomiting and hiccup were present, and the value for blood-urea rose to 304 mgrm. per 100 c.c. The patient's condition then improved, and by the sixth day the value for blood-urea had fallen to 100 mgrm. per 100 c.c. On the twelfth day he collapsed very suddenly and died within a few moments.

An autopsy was not obtained. Histologically the fold consisted of loose and vascular fibrous tissue, covered by transitional epithelium.

COMMENT

In both cases, and especially in *Case* 2 as a result of the estimations of bladder pressure, it was thought at first that the urinary retention was an expression of neuromuscular dysfunction, which may exist in the absence of somatic neurological signs. In the final analysis it is clear that in both cases only mechanical obstruction was present.

In both cases the position and the structure of the valve-like folds were identical. Their histological appearance does not give any clue to their origin. It has been suggested to one of us (J. R. L.) by Dr. John Beattie, Conservator to the Royal College of Surgeons of England, that they may be a result of the tendency to valve-formation seen in other muscular tubes (e.g., the gut) at levels where epithelium lining different embryological divisions of the tube becomes continuous. In our cases the valves appeared where the allantoic portion of the lower urinary tract becomes continuous with the Wolffian.

The possibility of the presence of this rare condition lends additional emphasis to the rule that a urological investigation, especially in children, should be complete. Only by strict adherence to this rule will cases of obstruction be recognized at a time when sufficient renal function remains to ensure that appropriate surgical treatment will be followed by permanent benefit.

One of the authors (K. H. W.) is indebted to Professor Ramsbottom and Mr. Charles Roberts, of the Manchester Royal Infirmary, for their kind permission to record *Case* 2.

REFERENCES

[1] HARRIS, A., "Congenital Vesical Neck Obstruction in a Female Child due to Cup-valve Formation: Open Operation: Complete Recovery", *Amer. Jour. Surg.*, 1933, xx, 64.
[2] CAMPBELL, M. F., "Trigonal Curtain Obstruction of the Bladder Outlet", *Jour. of Urol.*, 1932, xxvii, 157.
[3] WALLACE, W. J., "Unusual Bladder Obstruction", *Jour. of Urol.*, 1926, xv, 325.
[4] WATKINS, K. H., "The Clinical Value of Bladder Pressure Estimations", *Brit. Jour. Urol.*, 1934, vi, 104.

AN ANTERIORLY PLACED RENAL PELVIS

By W. K. IRWIN

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THE following case in which the pelvis of the kidney was displaced to the anterior surface instead of being situated at the mesial border seems worthy of record.

HISTORY.—Mr. K., aged 37 years, gave a history of pain in the left side of the abdomen extending over a period of three weeks. The pain, which was present all the time, showed itself as a dull ache or in the form of agonizing paroxysms radiating down the line of the left ureter and along the urethra. The patient also complained of urgency and frequency of micturition, nausea, and vomiting.

ON EXAMINATION.—There was tenderness in the renal area of the affected side, the urine was free from infection, and X-ray examination failed to produce

FIG. 628.—Pyelogram showing calices of left kidney directed forwards and medially. Right kidney normal.

FIG. 629.—Instrumental pyelogram of left or abnormal kidney.

any evidence of urinary calculus. By excretion urography (*Fig.* 628) the pelvis and calices of the right kidney were seen to be normal, while the calices of the left kidney were shown to be directed forwards and medially. *Fig.* 629 is an instrumental pyelogram of the left or abnormal organ.

FIG. 630.—Drawing of excised kidney.

OPERATION (Jan. 10, 1934).—Through a lumbar incision I exposed the left kidney, which presented an enlarged anteriorly placed pelvis. Further examination failed to demonstrate anything which might cause obstruction to the pelvic outflow, and a nephrectomy was done. The patient made an uninterrupted recovery and has remained well since the operation.

ANOMALIES OF THE REMOVED KIDNEY.—The organ was $5\frac{1}{2}$ in. in length, $2\frac{3}{4}$ in. in breadth, and $1\frac{1}{4}$ in. in thickness. The renal pelvis, as shown in the accompanying drawing (*Fig.* 630), was centrally placed on the anterior surface. As it passed upwards the pelvis separated into two primary divisions—a smaller upper and a larger lower branch. The upper branch separated into two sub-divisions and the lower into three sub-divisions before entering the hilum.

CALCULUS IN AN ECTOPIC KIDNEY

By R. TREVOR JONES
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C. B., aged 16 years, was admitted to Redhill County Hospital, Edgware, for investigation of a recurrent left-sided lower abdominal pain of five years' duration.

On Examination.—No abnormality was revealed by abdominal examination. The site of the pain was indicated as low down in the left iliac fossa. Dysuria and increased frequency were complained of during the attacks. Urine examination revealed scanty red blood-cells and a few pus cells.

Plain radiograms revealed nothing in the renal areas, but a large round shadow in the sacral region. Cystoscopic examination revealed nothing abnormal, the bladder and ureteric orifices were normal. Uroselectan pyelography revealed a normal right kidney and complete absence of any kidney in the left loin. The

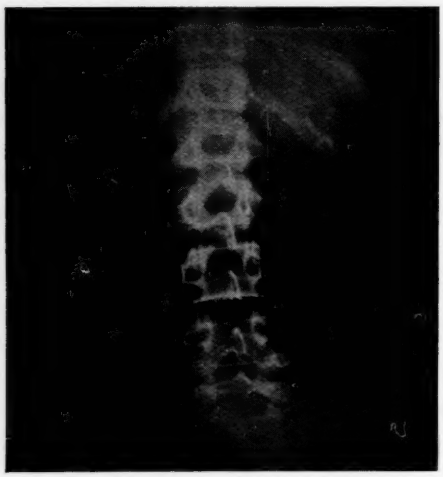

Fig. 631.—Skiagram taken three minutes after injection of uroselectan. Absence of any shadow in left renal area.

Fig. 632.—Skiagram demonstrating stone in ectopic kidney.

shadow below the promontory of the sacrum was now obscured by the shadow of a hydronephrotic ectopic kidney lying in the hollow of the sacrum. Confirmation of this was obtained by passing an opaque bougie along the left ureter and X-raying it. (*Figs.* 631–634.)

The urine obtained from the ectopic kidney was infected with streptococci.

Operation.—The abnormal kidney was removed through a left paramedian incision, retraction of the left rectus muscle, and division of the transversalis fascia, the peritoneum being stripped from the iliac fossa until the aorta and the left common iliac artery were exposed. The ectopic kidney was exposed by tearing through its fatty capsule. The blood-supply of the kidney was by numerous branches from the left and right common iliac arteries, which could be ligated

individually as they entered the upper border of the disc-shaped kidney. The vessels having been ligated, the kidney was withdrawn from the wound and the pelvis and ureter isolated. The ureter was traced to its attachment to the bladder, where it was divided and carbolized.

Convalescence was uneventful.

FIG. 633.—Pyelogram of ectopic kidney ten minutes after injection of uroselectan.

FIG. 634.—Skiagram showing catheter in left ureter.

FURTHER REPORT ON A CASE OF TRIGEMINAL NEURALGIA

BY J. HAMILTON BARCLAY

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IN a previous number of the BRITISH JOURNAL OF SURGERY (1921, ix, No. 34, 306) I reported "a case of trigeminal neuralgia in a boy, aged 10 years, treated by intracranial division of the second and third divisions of the nerve".

I now wish to report the subsequent history of this case because I think the after-history is interesting and instructive, and further because I believe that at the time I published the case in 1921 the patient was the youngest authentic case of trigeminal neuralgia recorded (10 years).

The operation was performed at the Hospital for Sick Children, Newcastle-upon-Tyne, on Feb. 6, 1920. The second and third divisions of the trigeminal nerve on the right side were divided intracranially. The pain was immediately relieved and remained so for eleven years (till the early part of 1931), when the patient commenced to have twinges of pain in the lower part of the right side of his face. The pains got worse and he came to see me at the Royal Victoria Infirmary, Newcastle-upon-Tyne. There was no doubt the trouble had recurred in the third division of the nerve. I referred him to my colleague, Dr. F. J.

Nattrass, who injected the third division with alcohol in October, 1931. The pain was relieved, but recurred in May, 1933, and the patient again consulted me at the Royal Victoria Infirmary. He was having severe spasms of pain in the distribution of the second and third divisions of the nerve on the right side. With the concurrence of Dr. Nattrass I decided to divide the sensory root of the nerve. The patient was then 26 years of age and married.

OPERATION.—This was performed on June 15, 1933. Nembutal, gr. $1\frac{1}{2}$, was given by the mouth the night before, and the dose was repeated on the morning of operation, together with morphia, gr. $\frac{1}{6}$, and atropine sulphate, gr. $\frac{1}{150}$. Open ether was administered and the anæsthetic was assisted by local infiltration with 1 per cent novocain. At operation the old temporal scalp-flap was turned down and the edges of the gap in the skull were exposed. The dura mater was firmly

FIG. 635.—Front and side views of patient's face at present time.

adherent to the edges of the gap and was freed with some difficulty. The dura and brain were lifted up from the middle fossa of the base of the skull. The foramen spinosum, foramen ovale, and foramen rotundum were easily exposed and recognized, and by further elevation of the dura mater the Gasserian ganglion was exposed lying in the cavum Meckelii. The dura was incised and the sensory and motor roots were cut across—as the third division carrying the motor fibres of the nerve had been divided at the first operation it was not considered necessary to differentiate between the motor and sensory roots. The dura and brain were allowed to fall down into position and the flap was sutured, a tissue drain being left in for a couple of days. The eye was covered up with an aseptic dressing to guard against possible corneal ulceration.

The operation was easier than anticipated. Some adhesions and fibrous tissue after the first operation were encountered, but did not constitute a formidable obstacle. There was at times some rather troublesome hæmorrhage, mainly venous,

but this was easily stopped by the coagulating diathermy current and by gauze pressure.

AFTER-HISTORY.—The day after the operation the patient was very fit and there was complete anæsthesia in the distribution of the trigeminal nerve on the right side. Recovery was smooth and uneventful; he was allowed up at the end of a week, and went home the following day. When last seen on Sept. 10, 1934, he was perfectly fit and absolutely free from any pain (*Fig.* 635).

HETEROTOPIC BONE FORMATION ASSOCIATED WITH ADENOCARCINOMA IN AN ABDOMINAL SCAR

BY ALFRED CLARK

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ALTHOUGH heterotopic bone formation in an abdominal scar is a recognized condition, it is relatively uncommon. The points of interest in this case were the presence of both bone and adenocarcinoma in the abdominal scar.

HISTORY.—The patient, a woman aged 51 years, was admitted to the Glasgow Royal Infirmary in August, 1933. At the operation the abdomen was opened by

FIG. 636.—Photomicrograph of section of mass, showing presence of bone and adenocarcinoma.

a right paramedian incision and a large inoperable carcinoma of the pelvic colon was found; the paramedian incision was closed and a colostomy was made. Six months later the patient was readmitted to hospital with a hard mass, about the size of a walnut, situated in the paramedian scar. The mass was excised and, after decalcification, sections were cut. The photomicrograph, *Fig.* 636, shows the

presence of bone and adenocarcinoma, and *Fig.* 637 is a higher-power magnification of a part of the bone.

Commentary.—The presence of the adenocarcinoma in the scar was probably due to implantation of cells taking place at the first operation, and this itself is rare. According to Leriche and Policard the factors necessary for the formation

FIG. 637.—A portion of the bone observed under high-power magnification.

of bone are an ossifiable medium, an excess of calcium, and an adequate blood-supply, and it is the presence of these requisite factors that permits the development of bone in abdominal scars and other tissues. In this case it is possible that the presence of the adenocarcinoma in the scar accounted for the adequate blood-supply, and thus permitted the development of the bone; on the other hand, the bone formation may have been independent of the adenocarcinoma.

I am indebted to Mr. Donald Duff for permission to publish this case.

ENDOSTEAL FIBROMA OF THE FIBULA

By D. F. CAPPELL
PROFESSOR OF PATHOLOGY, UNIVERSITY OF ST. ANDREWS

With the exception of enchondromata and the exostoses, both of which are commonly multiple and may be familial, simple tumours of the long bones are comparatively rare. The following case, therefore, is worthy of record on account of the rarity of the condition and the consequent difficulty in pre-operative diagnosis.

History.—C. D., aged 32, a dock labourer, was admitted to the Western Infirmary, Glasgow, on Feb. 4, 1928, under the care of Mr. Roy Young, complaining of a swelling in the right leg. In 1917 he had suffered a bullet wound in the right calf; this healed well, and so far as he knew the bones of the leg were not involved. About five or six years before admission he first noticed a swelling in the right calf just above the scar of the old wound. This had increased gradually in size, but had remained painless until two weeks before admission, when it became much larger and painful after a minor injury. The patient's general health was excellent and the family history good.

On Examination.—A large, firm, non-fluctuant swelling was found in the right calf, which measured 14 in. in circumference as compared with 11 in. on the left side. The growth was fixed to the fibula, but did not involve the tibia. There was no restriction of mobility in the limb. There was no apparent enlargement of glands; the Wassermann reaction was negative and the urine normal. X-ray examination revealed a large tumour mass involving the centre of the shaft of the fibula, the cortex of the bone being expanded, thinned, and at one point ruptured with extension of the tumour mass between the adjacent muscles. The growth appeared to have begun in the medulla and to have subsequently extended through the compact bone. A transverse fracture of the fibula was present (*see Fig.* 638). A few small areas of calcification were noted in the extrafibular tumour, but there was no evidence of bone formation apart from a layer of reactive periosteal bone on the surface of the intramedullary portion.

Operation.—Although the clinical and radiographical features of the growth did not definitely point to osteogenic sarcoma, it was considered advisable to obtain permission for amputation, but, in view of the doubt as to the malignant nature of the growth, it was decided to carry out microscopic examination of the tumour at the operation. Accordingly, the growth was exposed by Mr. Roy Young and portions from different areas were sectioned immediately and examined by Terry's method (1927). In the portions examined the tumour appeared to be simple in nature, and consequently local excision was performed, the fibula being cut through above and below the growth, which was then separated from the surrounding muscles. A large branch of the posterior tibial nerve was found to pass into the tumour and had to be sacrificed during the removal.

After-history.—This was uneventful. For some time difficulty in walking was experienced, but after three months this had practically disappeared and an excellent functional result had been obtained. The patient last reported on Oct. 2, 1934, and was then found to be in excellent health and able for full work as a dock labourer.

PATHOLOGICAL REPORT.—The tumour consists of two main portions, that within the fibula and that external in the surrounding tissues. The naked-eye appearance of the specimen after sectioning in the coronal plane is shown in *Fig.* 639. The mass within the fibula measures 7 × 2·5 × 2·5 cm.; it fills the interior of the medullary cavity and has produced expansion and thinning of the compact cortex. In most places the tumour mass is covered by a thin shell of dense bone, apparently newly formed during the gradual expansion of the shaft of the fibula. The bossy surface of the growth is shown also by the irregular shape of the expanded bone as seen in the X-ray (*Fig.* 638). On the postero-mesial aspect of the fibula

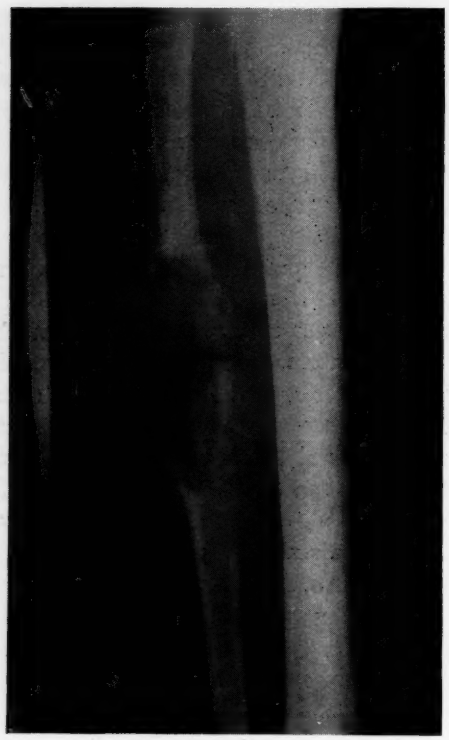

FIG. 638.—Lateral X-ray photograph. The plates show the expansion of the fibular shaft with the formation of a new layer of periosteal bone on its surface. Scattered foci of calcification are seen in the external tumour medial to the fibular shaft.

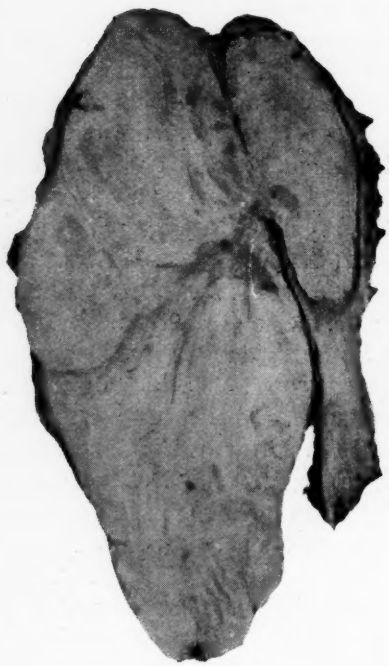

FIG. 639.—Coronal section of the resected fibula and tumour showing the fasciculated appearance of the cut surface with small areas of calcification. Note the expansion of the fibular shaft. (× ½.)

the bony cortex has disappeared and the intramedullary growth is joined to the larger external mass by a pedicle 2 cm. in width. The external mass, measuring 16 × 7 × 6 cm., is of more or less ovoid shape, with a fairly smooth bossy surface, and it projects backwards and inwards from the fibular shaft to occupy the calf of the leg. On section the tumour presents an almost uniform glistening whorled appearance resembling white fibrous tissue. The actual tumour tissue appears to be separated from the muscles by a well-defined capsule and is sharply delimited from the medullary cavity and bony cortex, the architecture of which is unaffected except in the immediate vicinity of the growth. A few small irregular nodules of hyaline appearance are seen near the bony attachment; some of these are intensely

calcified, but there is no true bone formation. Areas of cystic degeneration are not present, and the tumour is devoid of areas of pigmentation or hæmorrhage. One half of the growth was preserved as a museum specimen; the other half was thoroughly sectioned, but failed to reveal any areas of more cellular structure than those here shown (*see Figs.* 640, 641).

The structure of the growth is not suggestive of neurofibroma, and the inclusion of a large branch of the posterior tibial nerve appears to be incidental. The evidence is in favour of the intramedullary origin of the tumour, and, while the growth arose in the vicinity of the old bullet wound, there is no evidence that it was in any way related to the former injury.

Microscopic examination of numerous portions of the tumour reveals a fairly uniform structure, the growth being composed of whorled bands of elongated spindle-shaped connective-tissue cells separated by an abundant intercellular collagenous stroma (*Fig.* 640). At its margin the tumour is well encapsulated,

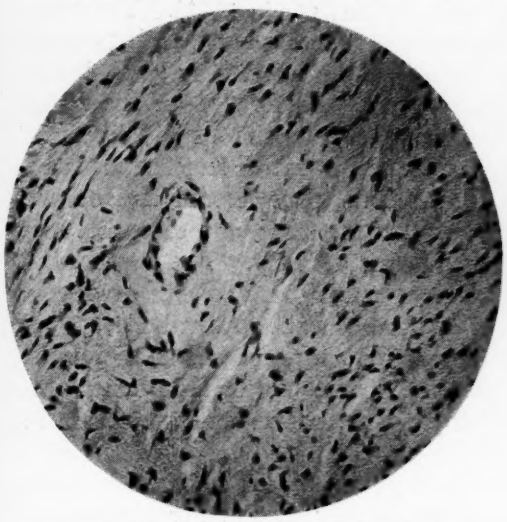

FIG. 640.—The greater part of the tumour is composed of rather acellular white fibrous tissue, in which occur a few blood-vessels with thick hyaline walls. (× 150.)

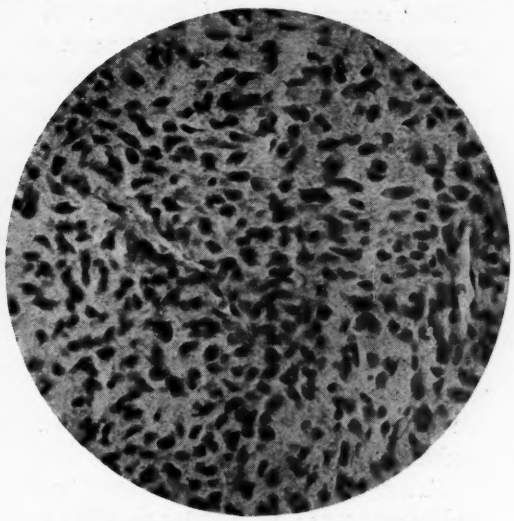

FIG. 641.—The most cellular area found after extensive microscopic examination of one half of the tumour. The cells tend to be stellate, and are separated by a soft mucoid matrix. The vessels are more numerous and are thin-walled. (× 250).

the structure thus being that of a moderately hard fibroma. In other parts the structure is more cellular, the individual elements being stellate rather than fusiform, and the intercellular matrix of looser texture. In a few places the stellate cells are separated by a mucoid matrix, but such myxomatous areas are small and not of frequent occurrence. There is no trace of osseous or osteoid tissue throughout the growth, and the matrix is everywhere of collagenous type. Giant cells of osteoclast type and phagocytes containing altered blood pigment appear to be completely lacking. The structure of the bone above and below the growth appears to be normal, and fibrous transformation of the marrow is quite absent. The blood-vessels generally present thick, rather hyaline walls, but a few thin-walled vessels are found in the more cellular areas (*Fig.* 641).

The general structure thus suggests a tumour of simple fibrous nature, or of, at most, a very low grade of malignancy; in view of the favourable outcome of the case, it is proposed to classify the tumour as an endosteal fibroma.

COMMENTARY

The interest of this case lies in the rarity of the condition and in the difficulty of pre-operative diagnosis. The long duration without noteworthy disability, the very gradual increase in the size, and the absence of pain until a minor injury led to pathological fracture, all suggested that the growth was not an osteogenic sarcoma. The site of the tumour in the centre of the fibular shaft is unusual, and the radiographic appearances were not typical of any of the commoner tumours or other lesions of bone.

In the differential diagnosis the localized form of osteitis fibrosa might require consideration. In this disease the onset is usually in early life, and its first appearance at the age of 26 years would be exceptional. Cystic change in the new-formed fibrous mass is the rule in the generalized form of osteitis fibrosa associated with hyperparathyroidism, but it is less common though not unknown in focal osteitis fibrosa (Knaggs, 1926; Elmslie, Fraser, et al., 1933). The latter authors record a case of widespread osteitis fibrosa not associated with hyperparathyroidism in which there was fusiform expansion of the left fibular shaft, but in addition changes were present in many other bones. Exploration of the femur revealed dense but highly vascular fibrous tissue at the site of old fractures. Throughout this fibrous mass were small areas of bone without true lamellar structure. In osteitis fibrosa, while the new-formed fibrous mass may distend and destroy the bony shaft, it does not as a rule spread beyond the shaft, and extension in a tumour-like fashion between neighbouring muscles is unlikely. The new-formed fibrous tissue in localized osteitis fibrosa usually contains some osseous or osteoid tissue, and foci of giant cells are met with. The absence of trabecular bone, osteoid tissue, and giant cells in the fibrous growth considered above renders it improbable that the case was one of osteitis fibrosa. Accordingly, we are of the opinion that the case is one of true neoplasm of relatively simple nature arising within the medullary cavity of the fibula.

The growth appears to belong to the group described as medullary fibrosarcoma by Ewing (1928), who states that the prognosis is relatively good and that metastases are unlikely, at least from the less cellular varieties. In the present case there is, after almost seven years, no evidence of local or other recurrence, and the patient, now in perfect health, is able to earn his living as a dock labourer. The good prognosis given from microscopic examination during the operation, and the conservative treatment accordingly adopted, have thus been justified.

In a search of the literature no closely similar case has been found, that most nearly parallel being one of Le Conte (1921), who reported a simple fibroma of the fibula with islets of cartilage in a woman of 21. In this case also the tumour had grown slowly for some years; total removal, which involved sacrifice of the peroneal nerve, was successful, the patient being well at least two years later. In the older literature there are numerous references to fibroplastic tumours of the fibula, but in most instances either these are clearly osteogenic sarcomata, or the details are insufficient for proper identification. Wintrebert and Lardennois (1897) record the case of a man of 42 in whom a large slowly growing tumour developed from the lateral aspect of the fibula at the site of an old injury and spread out into the intermuscular septa, pushing aside the muscles; amputation was performed, but on microscopic examination of the new growth, Bezançon pronounced it to be of

simple nature, being composed superficially of well-formed collagenous fibrous tissue, and in the deeper parts of bony lamellæ and osteoid tissue. Laffan (1893) also recorded a tumour in the head of the left fibula in a man of 52. The growth arose at the site of an injury two years previously, pain was noted in the region four years before operation, and during this period the growth attained a diameter of about three inches. Amputation was performed, and after a stormy convalescence complicated by secondary hæmorrhage, recovery ensued, the patient being apparently well at least seven years after operation. The growth is referred to as a 'sarcoma', but no mention of its structure is made.

None of the above cases is completely parallel to our own, in that the first two showed an admixture of other tissues, e.g., cartilage, osteoid tissue, and bone, while in the third the structure is not described in sufficient detail to make comparison of value. An association with the site of a former injury is noted in the second and third cases as in our own, but the significance of this is not clear.

Accordingly it would appear that simple endosteal fibrous tumours of the fibula and other long bones are exceedingly uncommon. The present account is therefore desirable in order to put on record the clinical and pathological features of such a case, and the satisfactory outcome of local conservative treatment.

SUMMARY

An account is given of a case of endosteal fibroma of the fibula, in which the tumour had burst through the bony cortex and pushed aside the muscles. Partial resection of the fibula together with the extrafibular tumour was performed with satisfactory result, the patient being well and active without recurrence six years later.

I am indebted to Mr. Roy Young, Visiting Surgeon to the Western Infirmary Glasgow, for the clinical notes and X-ray plates of the case.

BIBLIOGRAPHY

ELMSLIE, FRASER, DUNHILL, NIK, HARRIS, and DAUPHINÉE, *Brit. Jour. Surg.*, 1933, xx, 479.
EWING, *Neoplastic Diseases*, 1928.
KNAGGS, *Inflammatory and Toxic Diseases of Bone*, 1926.
LAFFAN, *Med. Press and Circ.*, 1893, 499.
LE CONTE, *Ann. of Surg.*, 1921, lxxiv, 794.
TERRY, *Jour. Pathol. and Bacteriol.*, 1927, xxx, 573.
WINTREBERT and LARDENNOIS, *Bull. de la Soc. Anat.*, 1897, lxxii, 794.

REVIEWS AND NOTICES OF BOOKS

Modern Operative Surgery. Edited by G. GREY TURNER, M.S., F.R.C.S., F.A.C.S. (Hon.), Senior Surgeon, Royal Infirmary, Newcastle-upon-Tyne, etc. Second edition. In two volumes. Medium 8vo. Pp. 1764, with 860 illustrations in text and 19 plates. 1934. London: Cassell & Co. Ltd. £3 3s. net.

THIS work was originally planned and edited by the late Mr. Herbert Carson. The first edition was published in 1924. Mr. Carson died while arrangements were being made for the production of the second edition, and the editorship was undertaken by Professor Grey Turner.

The general plan of the book has not been altered, though new sections have been added on radium by Mr. Geoffrey Keynes and on the surgery of the sympathetic system by Mr. Geoffrey Jefferson. The second edition contains 200 more pages and nearly 140 more illustrations than the first, and the extra space has been adequately employed in presenting the improvements in surgical knowledge and technique which the intervening ten years have afforded. The influence of the editor is felt widely throughout the book and is characteristically expressed in an introduction, which compresses the experience of a close observer into a few pages of friendly and broad-minded advice.

One of the chief features of this work is the discussion of the indications for the various operations described, a discussion which includes the choice of anæsthetic and the after-treatment of the patient. The argument as to time and type of operation is perhaps seen at its best in the article on appendicitis.

Limitations of space make it necessary to reduce the descriptions of many operations to a short outline and to omit a discussion of their indications, but the value and scope of the book has been greatly increased in many, though not in all, of the contributions, by the addition to the text of references to the original accounts given by their introducers. The second edition of *Modern Operative Surgery* has fully maintained the tradition of the first, and by amplifying its scope has increased its value to the present generation of surgeons.

Aids to Operative Surgery. By CECIL P. G. WAKELEY, D.Sc. (Lond.), F.R.C.S. (Eng.), F.R.S. (Edin.). Second edition. 6¼ × 4 in. Pp. 225 + viii, with 3 illustrations. 1934. London: Baillière, Tindall & Cox. 3s. 6d. net.

WE confess that we took up this little book with the biased feeling that it could not be worthy of much attention; but we are glad to say that a careful perusal at once altered our opinion. It is remarkable how the author has succeeded in including so much of the essentials of his subject within so small a compass and yet how clearly he has described the steps of the operations and their indications. For example, under 'Operation upon the Autonomic Nervous System' the various modern procedures of sympathectomy, ganglionectomy, and ramisection are excellently summarized in four pages. It is a great achievement and should prove very useful for rapid reference.

Applied Anatomy: the Construction of the Human Body considered in Relation to its Functions, Diseases and Injuries. By GWILYM G. DAVIS, M.D., Late Professor of Orthopedic Surgery and Associate Professor of Applied Anatomy in the University of Pennsylvania. Revised by GEORGE P. MULLER, M.D., Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania, and five Associates. Ninth edition. Large 8vo. Pp. 717 + xii, with 674 illustrations. 1934. London: J. B. Lippincott Company. 42s. net.

THIS is certainly one of the most comprehensive and best illustrated text-books on applied anatomy which we know, and we welcome this new and enlarged edition. The number, accuracy, and artistic beauty of the illustrations are alone well worth the purchase of the book.

Its main feature is the close correlation of surgical procedures to the underlying anatomical structures of the body. In the present edition the sections on the abdomen, gynæcology, urology, and the brain and spinal cord have been entirely re-written, whereas those dealing with the limbs remain substantially the same as in former editions. Our only criticism is that the description of the anatomy and surgery of the sympathetic nervous system is very incomplete. We think that the anatomy of the approach to the cervical and abdominal cords and ganglia should be fully described in a work of this importance.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE MCGREGOR, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of the Witwatersrand; Assistant Surgeon, Johannesburg General Hospital. With a Foreword by Sir HAROLD J. STILES, K.B.E., F.R.C.S. (Edin.). Second Edition. Crown 8vo. Pp. 644 + xx, with 639 llustrations. 1934. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

WE are not surprised that a second edition of this remarkable and most useful book has appeared so soon. It is packed from first page to last with most useful information and the illustrations are models of diagrammatic clearness. The anatomical basis of surgical procedures is set out in a way which is easily understood and remembered. For example, the anatomy of and many modern operations on the sympathetic nervous system may here be studied in concise and accurate presentation. The section on amputations has been entirely re-written, so as to bring it into line with modern ideas about late results and artificial limbs.

Visceriti e Perivisceriti digestive addominali croniche. By FRANCESCO ZAGARESE. Crown 4to. Pp. 359, with 60 illustrations. 1934. Bologna: Nicola Zanichelli. L. 40.

IN a preface to this work of his sometime pupil's, Professor Paolucci says that it is something more than an eclectic exposition of the manifold theories and opinions on the subject, it is "a complete and original study, that brings a contribution of the first importance to a difficult problem." He adds, a little naïvely, that he is particularly grateful because he finds therein all the promptings of his own teaching, all his own opinions, all the diagnostic and therapeutic criteria relied upon in his clinic. No doubt this is true, too, but the author has not confined himself to the work of his own school—indeed, it is very clear that his work in Paris with Pauchet has greatly influenced him—and he has brought a critical and sensible mind to bear on a question that does not leave much room for originality.

It is satisfactory to find that he is not enthusiastic about any mechanical means for dealing with or preventing adhesions. The one clear dictum is this: there is only one thing the surgeon can do for the relief of symptoms due to perivisceritis, and that is to eliminate completely the originating focus of infection. He holds that once this focus (which is easily and often overlooked) is removed, adhesions and the like take care of themselves.

There is a bibliography, mainly Italian and French.

Chirurgie du Pancréas. By P. BROCQ (Paris) and G. MIGINIAC (Toulouse). Large 8vo. Pp. 428, with 74 illustrations. 1934. Paris: Masson et Cie. Fr. 75.

THIS is a comprehensive and thoroughly up-to-date survey of pancreatic surgery. Of the 423 pages of descriptive text, 114 are allotted to acute pancreatitis, 110 to chronic pancreatitis, and 49 to cancer of the pancreas—the three most important of the pancreatic lesions.

The first section is concerned with the various traumatic conditions of the gland. Owing to its deep and well-protected situation, *wounds* of the pancreas are recognized only during laparotomy or at autopsy, and many are overlooked owing to insufficient exposure of the gland. Reference is made to the three routes of exposure: (1) through the gastro-hepatic omentum, (2) through the gastro-colic omentum, and (3) by the 'inter-colo-epiploic' route, whereby the transverse colon and its mesocolon are separated from the omentum along the plane of fusion which occurs between them during embryonic life. Subcutaneous injuries—*contusions, lacerations, and ruptures*—are recognizable clinically at one of three periods: (1) immediately, when the associated shock and hæmorrhage necessitate laparotomy, (2) some days after the accident when there has formed an encysted hæmatoma of the lesser peritoneal sac, and (3) some weeks later when a traumatic pseudo-cyst of the lesser peritoneal sac has developed.

The section on *acute pancreatitis*, written by P. Brocq, is based upon 468 collected cases in all of which the diagnosis was verified either at operation or autopsy. A fundamental distinction is made between 'aseptic' cases, where the peritoneal effusion is sterile, and 'septic'; the former are the more frequent (386 aseptic to 82 septic). The pathological anatomy and histological changes in the various types of acute pancreatitis are well described and illustrated, and attention is drawn to the frequency with which old-standing histological lesions are met with—sclerosis, arteritis, phlebitis, catarrh of ducts: this indicates that it is on a previously damaged gland that acute pancreatitis has supervened, and is in keeping with the clinical observation that slight attacks often precede the final crisis of acute pancreatitis. The authors have carried out a large number of experiments upon dogs, and their researches and those of other workers have shown that ligature or section of the duct of Wirsung does not produce acute pancreatitis, but that this condition will follow injection into the duct of Wirsung of bile, of duodenal contents, of succus entericus, of microbic and other toxins, and of a 20 per cent solution of calcium chloride. Acute pancreatitis is stated to be primarily an 'auto-digestion' of the gland: the conditions in which activation of the pancreatic ferments is brought about are fully considered and special stress is laid upon the frequent association of acute pancreatitis with disorders of the biliary passages. Early operative treatment is advocated as the only safe course to adopt, and the recent tendency with some surgeons to avoid operation during the period of acute crisis is discountenanced. The essential operative procedures are fully detailed and some useful hints on post-operative management are given. It is estimated that diabetes develops in about 10 per cent of recoveries from acute pancreatitis.

The difficulty of estimating the frequency of *chronic pancreatitis* is noted since it is not characterized by any pathognomonic sign, may remain unrecognized throughout life, and is, moreover, frequently overlooked during the course of abdominal operations. The authors stress the necessity of examining the pancreas in all laparotomies for affections of the stomach, duodenum, and bile-passages. Many troubles that remain unrelieved after cholecystectomy are due to an overlooked associated chronic pancreatitis. There is a full description of the clinical manifestations of chronic pancreatitis and of the laboratory methods of studying the functional state of the gland with regard both to its external and its internal secretions. Compression troubles, while affecting chiefly the biliary tract, may rarely concern the pyloro-duodenal region and produce food-stasis, or by pressure upon the solar plexus may cause attacks of pain which may be so severe as to simulate the gastric crises of tabes. An excellent account is given of the pathological anatomy of chronic pancreatitis and of the associated changes in the biliary passages and adjacent viscera, while a series of fine illustrations of microscopical sections of the various types of chronic pancreatitis adds very materially to the value of the work. Surgical treatment is advocated whenever there is evidence of compression, and the indications for the various operative procedures that may be called for are most judiciously stated.

In the section dealing with *cancer of the pancreas* reference is made to the great difficulty of distinguishing between cancer and chronic pancreatitis whether clinically, or during laparotomy, or at autopsy; histological examination is essential, but a caution is given against lightly undertaking biopsy during laparotomy owing to the risks of post-operative hæmorrhage and of auto-digestion of the gland. In no other region can cancer produce such rapid wasting of the body as when situated in the pancreas. The islands of Langerhans usually escape, so that glycosuria is only rarely met with. The symptoms and physical signs receive separate description according to whether cancer affects the head, body, or tail of the gland, and there is a very full discussion of the diagnosis of obstructive jaundice. Cholecyst-gastrostomy is advised for the relief of jaundice, and where this is not feasible, either because of the absence or disease of the gall-bladder or because of the patient's weak general state, the bile should be drained externally either by cholecystostomy or choledochostomy.

Contrary to the opinion of many writers who consider that most *cysts of the pancreas* are of neoplastic origin, it is here stated that of all the fluid swellings of pancreatic origin the most frequent are the 'necrotic' pseudo-cysts arising as a sequel to an acute pancreatitis.

Other special chapters deal with syphilis, tuberculosis, pancreatic calculus, pancreatic fistula, and anomalies of the pancreas.

This volume is a veritable mine of information, and is likely to remain for a long period the standard work of reference in connection with the surgery of the pancreas.

Sincronización quirúrgica: Team Standard Operatorio sincronizado: Apendicectomía. By Dr. GUILLERMO BOSCH ARANA (Buenos Aires). 10½ × 7 in. Pp. 215, with 212 illustrations. 1934. Buenos Aires: "El Ateneo".

THIS remarkable book suggests the reduction of a surgical operation—in this case appendicectomy—to a rigid formula, which prescribes for each person engaged in its conduct his exact individual movements. All actions are precisely synchronized and are performed without a word spoken. A table of the 'decalogue' for each participant—surgeon, first assistant, second assistant, sister, and so on, is set out. A multiplicity of pictures—212 in a book of 215 pages—makes meticulously clear the minutiæ of every detail, so that others may emulate the 'team standard' of the author, who is Professor of Surgical Technique in the Faculty of Medicine of Buenos Aires.

Two questions force themselves upon the reader. "Will human nature stand the strain?" and "Is it worth while?" The author's obvious enthusiasm and high ideals might almost persuade the young to answer, "Yes!"

The Management of Fractures, Dislocations, and Sprains. By JOHN ALBERT KEY, B.S., M.D., Clinical Professor of Orthopedic Surgery, Washington University School of Medicine; and H. EARLE CONWELL, M.D., F.A.C.S., Orthopedic Surgeon for the Tennessee Coal, Iron and Railroad Company, Birmingham, Ala. Imperial 8vo. Pp. 1164, with 1165 illustrations. London: Henry Kimpton. 63s. net.

IN this text-book the authors have given a very comprehensive account of American methods of treatment of fractures, dislocations, and sprains. Part I of the text-book consists of an account of the principles of fracture treatment, and the equipment necessary to carry these out in a hospital of 100 to 200 beds, followed by an account of their complications and the treatment of compound fractures. The end of Part I is occupied by a consideration of medico-legal problems and the Workman's Compensation Law as it affects fracture cases in America. Part II of the text-book is confined to the diagnosis and treatment of specific injuries.

The subject-matter is well arranged and excellently illustrated, but there are many views expressed which would not find general acceptance in this country. One is surprised to read that the authors favour the padded plaster cast, when the unpadded cast—though admittedly more difficult of application—secures immobilization in a way which can never be obtained with a padded plaster. Also spinal anæsthesia is not recommended for reduction of fractures of the lower extremity, although the authors admit they have little experience of it.

In the chapter on first-aid treatment of fractures, the authors, although appreciating the value of a Thomas splint, make the rather surprising statement that they are seldom available, and therefore describe the use of board splints. Also, in fractures of the spine the importance of transporting the patient in the prone position is not recognized and a cumbersome ladder splint is described. In the section on special injuries, fractures of the spine are dealt with in considerable detail. In reference to Kümmell's disease, the more generally accepted view that these cases are undetected compression fractures is taken, and a fusion operation is advocated. A considerable amount of space is also devoted to low back sprain and the methods of examination and treatment.

The classification of fractures around the elbow-joint adopted is rather a confusing one, and the authors only recommend the Jones sling as a convalescent dressing, when there is no doubt that many fractures around the elbow-joint can be treated from the very beginning in this way. Also an injustice is done to the teaching of the late Sir Robert Jones in the statement that he recommended treatment in acute flexion; this should be altered to "easy flexion". Many surgeons will also consider eight weeks too early 'to stretch' an elbow-joint, even in the absence of abnormal calcification shadows around the joint.

In the treatment of fractures of the forearm, anterior and posterior, wooden splints are employed and then incorporated in a light plaster cast, a method which must procure very imperfect immobilization and lead to trophic changes in the muscles of the forearm. In the treatment of fractures of the neck of the femur, the advantages of the Smith-Petersen nail are not emphasized although it is agreed that the operation is technically somewhat difficult. The statement that many patients are poor surgical risks leads one to the remark that these patients will tolerate just as badly a long period of fixation in plaster, which gives a poor percentage of union compared with the 'nailing' operation. Fractures of the femur in children

are also better treated by immobilization in a plaster spica, in preference to the older Bryant's method which is described.

We think that although this text-book may give an account of fractures and their treatment as practised in a large industrial centre in America, one cannot recommend it to the English reader, because so many of the methods of treatment described are such as would hardly find recognition in a modern fracture clinic in this country.

The Treatment of Fractures. By LORENZ BÖHLER, Director of the Hospital for Accidents, Vienna; Lecturer on Surgery in the University of Vienna. Translated from the Fourth Enlarged and Revised German Edition by ERNEST W. HEY GROVES, M.S., F.R.C.S., Emeritus Professor of Surgery, University of Bristol. Large 8vo. Pp. 578, with 1059 illustrations. 1935. Bristol: John Wright & Sons Ltd. 42s. net.

BRITISH and American surgeons will be grateful to Professor Hey Groves for his clear and readable translation of the fourth edition of Lorenz Böhler's book on the treatment of fractures. Although there is a valuable section on general principles, this work is in no sense a complete text-book, and is obviously not intended for the instruction of the undergraduate student or general practitioner. It is a special monograph presenting in considerable detail both the technique in vogue at the present time and the administrative arrangements of the Vienna Hospital for Accidents, which is now world-famous.

Böhler's earlier work on fractures of the carpus, fractures of the forearm, and other common types of injury has been fully expounded in previous editions, and his practice in this respect may be said to be stabilized. In the latest edition, which now runs to 578 pages, special consideration is devoted to fractures of the spine (41 pages), fractures of the upper end of the femur (39 pages), and fractures of the os calcis (24 pages). In crush fractures of the spine he has adopted the hyperextension method which is in general use in British clinics and which was many years ago first practised by Davis and others in the United States. In fractures of the femoral neck, without discarding the Whitman method of manipulative reduction and prolonged plaster fixation, Böhler has become an enthusiastic exponent of the operative fixation of the fracture by means of the Smith-Petersen three-flange pin. It would appear that he now favours the extra-articular operation of Sven Johannsen, driving in the nail after the preliminary insertion of two wire guides under X-ray control. Böhler's special addition to this technique is to use two sets of X-ray apparatus in order to cut down the time necessary to carry out the whole procedure, which, he states, takes not more than fifty to sixty minutes "in lucky cases"! He appears to be unduly apprehensive of the dangers of the original method of Smith-Petersen, in which a complete exposure of the fracture is a necessary step, but which, in the hands of the experienced surgeon, takes a comparatively short time.

The attempt to treat severe os calcis fractures on rational mechanical lines is perhaps Böhler's most notable technical contribution to the treatment of fractures. In this chapter, however, we miss the note of optimism which characterized his earlier efforts to deal with these difficult fractures. Also there is no statement regarding the true end-results in men who are called upon to return to the heaviest type of labour. What we want to know is how many of these individuals can climb ladders and walk about scaffolds.

The salient features of Böhler's teaching are as follows: The importance of early and accurate reduction, under X-ray control; the value of local anæsthesia; the efficacy of the unpadded plaster cast; the inadvisability of open reduction or fixation of any fractures, except those of the olecranon, patella, and neck of the femur; the importance of avoiding œdema by keeping the limb raised during the early stages of treatment, and by the use of Unna's paste for the lower limb in later stages; the treatment of all wounds by open exposure, without the use of any dressing; the danger of massage and passive movements when applied to recent fractures; the importance of active movements from the very first of all joints not actually fixed; the value of ambulant treatment for many fractures of the leg, especially Pott's; that the bad results in fractures are often due to errors in treatment, rather than to the original injury; the importance of team work, including unity of control and adequate follow-up; and finally the enormous economic value of systematic fracture treatment.

The appendix contains a convincing account of both the clinical and financial efficiency of the fracture organization which Böhler has established in Vienna. There can be no doubt that this monograph is an essential addition to the library of the fracture service of every large hospital in Great Britain and America.

L'Epaule : Anatomie des Formes extérieures, Anatomie radiographique, Chirurgie opératoire. By ANTOINE BASSET, Professeur agrégé à la Faculté de Paris ; and JACQUES MIALARET, Interne des Hôpitaux de Paris. Royal 8vo. Pp. 292, with 116 illustrations. 1934. Paris : Masson et Cie. Fr. 65.

THIS is one of a series of monographs on the joints, that on the knee having appeared already, and that on the elbow being in course of preparation. The anatomical and radiographical sections are very clear and well illustrated. The superposition of the muscles and ligaments over the outline bony skeleton shows many useful points and makes the comprehension of the joint mechanism simple. The radiograms are particularly valuable as giving the normal outline of the bones seen in various positions of the arm, e.g., the arm abducted and the X-ray tube placed in the axilla. Also the X-rays of the shoulder after bismuth injections of the blood-vessels and articular cavity are most instructive.

The chapters on the surgery of the joint are characterized by completeness and by a wealth of illustration. The number and variety of methods for each surgical procedure leave one a little bewildered. Simple methods are hardly touched on, whilst methods of great complexity are described at length. For example, the exposure of the shoulder by splitting the deltoid is much the easiest approach to the joint ; also a sling of fascia lata round the neck of the humerus is the ideal method of treating the recurrent dislocation, but neither of these methods finds a place. A great number of ingenious methods of treating the flail shoulder, including different muscle transplants, are described. The book is a very valuable storehouse of information, most useful for reference.

Operationstaktik bei Erkrankungen der Gallenwege. By W. M. STERN (Paris) and R. FOURCHE (Nancy). Translated by Dr. E. HAYWARD (Berlin). Super royal 8vo. Pp. 256 + viii, with 203 illustrations. 1934. Berlin and Vienna : Urban & Schwarzenberg. Paper covers, RM. 12 ; bound, RM. 13.50.

ONE is naturally intrigued by the origin and title of this book. For the work of two French surgeons to be translated and edited by a German is a certain evidence of its worth. Then there is the curious title of " The Tactics of the Operations on the Biliary Passages ". By this, we understand from the preface, is meant that not only is technique described, but also the circumstances of the local and general condition which determine the type and extent of each operation. The work is divided into three parts. The first deals with examination and preparation preliminary to operation (10 pages), the second with the details of various operations (206 pages), and the third with associated lesions, complications, and post-operative procedures (36 pages).

The feature of this book which makes it of the greatest possible value is the number and character of the illustrations. These are nearly all about three-quarter-life-size line drawings which, whilst of great artistic merit, are wonderfully clear and diagrammatic.

On Osteogenic Sarcoma. By JACOB VAN DER SPEK. Royal 8vo. Pp. 217, with 24 X-ray plates. 1933. Kemink en Zoon N.V., Overden Dom., Utrecht.

THIS is an excellent monograph in English on bone sarcoma in which a very good review of modern pathology, prognosis, and treatment is given. The chief original material is derived from forty-two cases observed in Noordenbos' clinic at Amsterdam. The chief value of the book consists in the fair and complete summary of modern views, both American and Continental, on the subject. The dangers and fallacies of biopsy as a method of diagnosis are fairly pointed out. These tumours are often of such varied structure that the examination of a small piece is of little value, so that in reality the whole tumour should be excised and sectioned before its nature can be known accurately. A just protest is made against the attitude of non-intervention in most cases. Although the proportion of five-year cures may be small, yet the prognosis is certainly better if radical amputation or disarticulation is done than if the case is left alone, and, moreover, there is diminution of pain and suffering. The last section of the book deals with giant-celled sarcoma. It is clearly proved that the operation of enucleation is unsatisfactory and should be abandoned for resection.

Head Injuries. By L. BATHE RAWLING, M.B., M.Ch. (Cantab.), F.R.C.S., Consulting Surgeon to St. Bartholomew's Hospital and to the West End Hospital for Nervous Diseases. Demy 8vo. Pp. 86 + vi, with 22 illustrations. 1934. London: Oxford University Press. 7s. 6d. net.

IN his preface to this book the author warns his readers that he intends to treat this highly important subject in a personal and dogmatic manner. In many ways this has produced an admirable result, and the opinions of an authority such as Mr. Bathe Rawling are necessarily weighty. The book seems rather too elementary, and the author slurs over some points on which we would have valued his opinions. Thus the indications for decompression in cases of cerebral irritation are summarily dealt with. It is on such a point that we should like to know Mr. Rawling's procedure. The treatment of persistent cerebrospinal fluid loss is also omitted.

The book gives a clear and easily readable account of the commoner skull fractures and the usual forms of cerebral damage. It is clearly paragraphed and reference is easy. We are in complete agreement with the author in condemning the routine administration of hypertonic intravenous salines in head injuries. Such exhibition should always be controlled by lumbar puncture and the clinical picture. The author rather deprecates the use of the spinal manometer as an adjunct to lumbar puncture. He tells us that frequent experience enables one to estimate the pressure. We feel that this is retrograde. Mistakes will occur while this degree of experience is being obtained. Loss of fluid while the surgeon estimates the pressure may be harmful. An astute clinician can estimate the blood-pressure by a finger on the pulse or the number of red cells by examination of the mucous membranes, but few would now dispense with the sphygmomanometer or the blood-count.

The need for frequent re-examination of the clinical and neurological state in the management of such cases could be more stressed, as changes are notoriously rapid. These criticisms are, however, minor, and the book will be read with profit by all who are called upon to treat cases of head injuries.

Diseases of Children. Edited by HUGH THURSFIELD, D.M. (Oxon.), M.A., F.R.C.P., Physician, Hospital for Sick Children, Great Ormond Street; Consulting Physician, St. Bartholomew's Hospital; and DONALD PATERSON, M.D. (Edin.), F.R.C.P., Physician to Out-patients, Hospital for Sick Children, Great Ormond Street; Physician in Charge of Diseases of Children, Westminster Hospital. With contributions by 36 authors. Third edition. Royal 8vo. Pp. 1152 + xii, with 277 illustrations. 1934. London: Edward Arnold & Co. 50s. net.

THE third edition of this standard work, whilst retaining its general arrangement and outlook, contains much that is new and revised, both in illustrations and text. New articles on blood transfusion, orthodontic treatment, diseases of the accessory nasal sinuses and the ear, and on cystoscopy and pyelography, have added breadth to the surgical aspects of the volume. In general, however, the book does not attempt to go deeply into the surgical problems of childhood, but rather to fit them into their appropriate place with a much wider medical background. Surprisingly, congenital pyloric stenosis has been delegated to a surgical contributor. Without criticism of the substance of the article, which is excellent, we doubt the wisdom of this, the operation being an incident only in a disease with pre- and post-operative periods complicated by problems essentially medical. The writer, we think, does not emphasize sufficiently the importance of early diagnosis as being *the* factor responsible for the lower mortality in private patients.

As one of the major emergencies in childhood acute osteomyelitis might well receive more attention and space to bring it into a proper perspective in a text-book essentially for the practitioner, yet one in which so much is devoted to the rarer forms of bone disease.

In spite of such minor criticisms, which are easy in a volume covering such a wide field, we feel the book is a most valuable compilation which will be widely read and appreciated.

Report on Seventh International Congress of Military Medicine and Pharmacy and Meetings of the Permanent Committee (Madrid, Spain, May 29–June 3, 1933). By CAPT. WILLIAM SEAMAN BAINBRIDGE, M.C.–F., U.S.N.R, Member of the Permanent Committee; Delegate from the United States. Large 8vo. Pp. 88. Menasha, Wis.: George Banta Publishing Co.

CAPTAIN BAINBRIDGE has again performed a valuable service in reducing to readable bulk the voluminous report of this International Congress. The subjects dealt with were as follows:

(1) General principles regarding medical services in war time: their application to the new rulings of the Geneva Convention. (2) Preventive vaccination in the Army, Navy, and Air Force. (3) Treatment in advanced posts of urgent surgical casualties in a war of movement. Scheme of a specialized formation: its technical organization and its employment from a tactical viewpoint. (4) Preserved foods as a regular ration for soldiers in peace time or in the field: their modes of preparation and analysis. (5) Comparative study of odonto-stomatological and administrative services in the different Armies, Navies, and Air Forces. (6) (Veterinary Section) Glanders: (*a*) Clinical and prophylactic study; (*b*) Bacteriological study; (*c*) Anatomo-histopathological study.

There is a short foreword by Rear Admiral P. S. Rossiter of the United States Navy. The little book is well printed and produced. Some errors in the names of the delegates are noted, accounted for by mistakes in the original proceedings.

Benign Encapsulated Tumours in the Lateral Ventricles of the Brain: Diagnosis and Treatment. By WALTER E. DANDY, M.D., Adjunct Professor of Surgery, Johns Hopkins University. Large 8vo. Pp. 189 + viii, with 83 illustrations. 1934. London: Baillière, Tindall & Cox. 20s. net.

THIS monograph is the outcome of an analysis of 15 cases of primary benign encapsulated tumours in the lateral ventricles of the brain. All were found at operation, and 14 totally removed. The mortality rate was low, only 3 deaths having occurred in the whole series. In addition to his own 15 cases, Dandy has been able to collect 25 cases from the literature, of which 23 were discovered at autopsy.

The importance of ventriculography is stressed at great length, and Dandy considers that when used correctly, such an investigation is without risk to life or function. It may be said that the only satisfactory treatment of these tumours is complete removal. The operative risk is reduced by the standard use of the electro-cautery, which has done so much to make all types of cerebral surgery safe. The other factors which matter are the use of avertin anæsthesia and continuous suction of the wound area.

The monograph, like all the works of Dandy, is a record of fine and critical observation and judgement, combined with superb operative technique. The illustrations are excellent and could not be bettered, while the analytical tables embodying every case will be found most useful.

Biological Flaps. By J. F. S. ESSER. Published by the Editor of the *Institut Esser de Chirurgie Structive*, Monaco.

ANYTHING from the pen of J. F. S. Esser must be most carefully studied by any reconstructive surgeon. When his epithelial inlay was first published in English in March, 1917, few of the surgeons who read it were alive to the immense possibilities that lay behind the thought. Similarly, to a modified extent, a certain type of antagonism was felt towards his rotation flap. The truth about these two procedures as practised by English-speaking races is that the ideas of both are eminently sound and when suitably modified of great practical value. It would seem that 'the ideal biological' outlook, as conceived by the inventor, has overshadowed in his mind the meticulous refinement that is demanded of the plastic surgeon in the United States or British Empire. Thus also does this new work, devoted to the subject of artery flaps, first strike a note of impracticability, but on reading deeper and taking it in conjunction with the transplantation of fingers and toes and the possibility of even larger limb replacements, one is forced to the conclusion that Dr. Esser has again struck a principle absolutely sound from the biologic point of view. The fact that many of the illustrated cases are poorly finished, or that they give evidence that similar results could be obtained by flaps not specially arterial in design, should not blind the reader to the immense value of this original contribution.

Take, for instance, the simple case of the war injury labelled 6D 39, 6D 40: the same flap could have been utilized with an ordinary pedicle based on the temporal artery; the pedicle returned to the scalp with no more scarring than exhibited in this case, and without the joining scar running down the patient's cheek which is necessary to embed the artery part of the pedicle in Esser's method. That the writer cannot detect any superior advantages of this

method over the established ones of the present day for the kind of injuries depicted in the book does not detract from the great value of the contribution or from its possibilities. The record of the work done as depicted in the book is indicative of the immense labour and ingenuity of the surgeon, and provides a monument of gratitude to him for the success with which these war wrecks have been converted into human beings.

The book is beautifully printed, and, considering the difficulties of obtaining records during the War, the illustrations are both profuse and clear, and there is no suspicion of the slightest dishonesty in the method of taking the photographs. One of the best restorations illustrated is that of L.1 to 5, but why he should be introduced twice complete within a few pages is not quite apparent. One of the most important contributions in the book is the stress which Esser places on the value of the venous and lymphatic supply as compared with the arterial. It has long been recognized that a flap dies through failure of the venous return rather than that of the arterial supply.

It may well be that the development of this biological artery-flap, when applied to restorations in which the donor site is on the body, will lead to a great simplification of these methods, and a great saving of time and money. Most particularly is one impressed with the need for experimentation to solve the question of cross-grafting from one patient to another, or even from a corpse. This problem would appear to be linked up with the gradual immunization of the tissues of the recipient to those of the donor. It is to be noted that the book is published by the Editor of the Institute Esser, which is an organization backed by the most scientific and influential people, having as its chief aim a non-political and international centre for reconstruction surgery. The establishment of such an institute will be an amazing tribute to the energy and imagination of Esser.

X-ray Interpretation. By H. CECIL H. BULL, M.A., M.B., M.R.C.P., Physician and Hon. Radiologist, Royal Waterloo Hospital, London. Demy 8vo. Pp. 382 + xxxiv, with 280 illustrations. 1935. London: Oxford University Press. 21s. net.

THE author has attempted to compress into some 370 pages nearly the whole of X-ray diagnosis, and a good deal of X-ray anatomy. Many of the simpler conditions are dealt with at length, such as fractures, so that the more difficult and complicated investigations are treated somewhat briefly. The chief value of the book, therefore, would appear to be for those requiring an introduction to radiology. The illustrations are all simple line drawings and silhouettes, and no reproductions of radiographs are shown. In such a section as that on the stomach this is no great disadvantage, but in lesions of the chest or bone diseases it is very difficult to understand the line drawings, unless one is already very familiar with the ordinary X-ray appearances.

The 1934 Year Book of Radiology. Edited by CHARLES A. WATERS, M.D. (Diagnosis), and IRA I. KAPLAN, B.Sc., M.D. (Therapeutics). Large 8vo. Pp. 512, with 454 illustrations. 1934. London: H. K. Lewis & Co. Ltd. 19s. net.

THIS book follows on the lines of the past volumes and gives an abstract (with references) of most of the important articles published in the past year. The numerous reproductions from the radiograms are clear, and the present volume, together with its predecessors, forms a valuable reference work.

Selecting a few of the abstracted articles of surgical interest, one might mention those on arteriography, an article describing some new experimental work on the intervertebral discs, and an investigation of the emptying of the gall-bladder after cholecystography. On the X-ray technique side mention is made of the demonstration of enlarged adenoids, and a vertical projection for the neck of the femur which may have considerable bearing on the treatment of fractures in this region. Many cases of bone diseases and tumours are quoted and a case of osteitis fibrosa cystica with a tendency to spontaneous cure. The X-ray examination of the colon in a case of Hirschsprung's disease before and after sympathectomy is also of interest.

Judging from the abstracts, more and more importance is being attributed to the pre-operative irradiation of malignant tumours, particular mention being made of breast cancer

and renal tumours. In this connection it is emphasized that wound-healing proceeds normally after properly applied radiation. Various auxillary methods of therapy in cancer are discussed, and the opinion is expressed that the beneficial effects of colloidal selenide therapy are chiefly due to the radiation used with it. Chronic inflammatory conditions are often benefited by radiation; of recent advances, the immediate relief and cure of post-operative parotitis by radiation should be useful to surgeons, but few would care to use it as the main measure in gas gangrene.

There is a full index of subjects and authors to facilitate quick reference to the various subjects. A perusal of this book should prove of use to the surgeon as well as the radiologist.

Füss und Bein: ihre Erkrankungen und deren Behandlung. By Prof. Dr. med. GEORG HOHMANN (Frankfurt a. M.). Second edition. Royal 8vo. Pp. 380 + x, with 326 illustrations. 1934. Munich: J. F. Bergmann. Paper covers, RM. 24; bound, RM. 25.80.

THIS book was originally written in 1923 and dedicated to Professor Fritz Lange on his seventieth birthday. It is a carefully written and well-illustrated book dealing with the anatomy, physiology and pathology of the foot, especially in regard to various deformities.

The section on gymnastics and physical treatment is very interesting as giving a well-ordered plan for massage and exercises applied for deformed feet. There is an elaborate account of various arch supports without very adequate discussion as to their rationale. In the treatment of various types of talipes and flat-foot great prominence is given to cuneiform osteotomies of the os calcis, navicular, and astragalus. In the treatment of hallux valgus, too, there are a great number and variety of operations described and discussed, chiefly cuneiform osteotomies and muscle transplants. We must admit to being a little bewildered by the complexity of these suggestions.

The so-called arthrosis and arthritis deformans of all the joints of the lower limb is figured and discussed. Many of the minor ailments, e.g., corns, ingrowing toe-nails, and varicose veins are included in this monograph, which certainly contains a great wealth of material and much subject for reflection and discussion.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Operative Surgery. By C. R. WHITTAKER, F.R.C.S. (Edin.), F.R.S.E. Fifth edition. Crown 8vo. Pp. 228, with 53 illustrations. 1934. Edinburgh: E. & S. Livingstone. 4s. 6d. net.

A Short Practice of Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, etc.; and R. J. MCNEILL LOVE, M.S. (Lond.), F.R.C.S., Surgeon, Royal Northern and Metropolitan Hospitals, etc. Second edition. Demy 8vo. Pp. 988 + viii, with 731 illustrations. 1935. London: H. K. Lewis & Co. Ltd. 30s. net.

Surgical Errors and Safeguards. By MAX THOREK, M.D., Professor of Clinical Surgery, Cook County Graduate School of Medicine, etc. Second edition. Large 8vo. Pp. 696 + xv, with 668 illustrations. 1935. London: J. B. Lippincott Co. 45s. net.

Clinical Pathology of the Jaws: with a Histologic and Roentgen Study of Practical Cases. By KURT D. THOMA, D.M.D., CHARLES A. BRACKETT, Professor of Oral Pathology in Harvard University, etc. Large 8vo. Pp. 643 + xii, with 423 illustrations. 1934. London: Baillière, Tindall & Cox. 38s. net.

Handbook of Anaesthetics. By J. STUART ROSS, M.B., Ch.B., F.R.C.S.E., Late Lecturer in Practical Anæsthetics, University of Edinburgh; and H. P. FAIRLIE, M.D., Anæsthetist to the Western Infirmary, the Royal Hospital for Sick Children, and the Dental Hospital, Glasgow. Crown 8vo. Pp. 299 + xv, with 66 illustrations. 1935. Edinburgh: E. & S. Livingstone. 10s. 6d. net.

Aids to Surgery. By CECIL A. JOLL, M.S. (Lond.), M.D. (Bris.), B.Sc. (Lond.), F.R.C.S., Senior Surgeon and Lecturer on Surgery to the Royal Free Hospital, etc.; and REGINALD C. B. LEDLIE, M.B., B.S. (Lond.), F.R.C.S., Surgeon to the Miller General Hospital, etc. Sixth edition. F'cap 8vo. Pp. 612 + x, with 44 illustrations. 1935. London: Baillière, Tindall & Cox. 7s. 6d. net.

Collected Papers of St. Mark's Hospital, London: including a History of the Hospital. Centenary Volume, 1835-1935. Compiled by the Medical Committee. Crown 4to. Pp. 440 + xxvi, with 96 illustrations. 1935. London: H. K. Lewis & Co. Ltd. 30s. net.

Surgical Diseases of the Chest. By EVARTS AMBROSE GRAHAM, A.B., M.D., F.A.C.S., Professor of Surgery, Washington University School of Medicine, St. Louis; JACOB JESSE SINGER, M.D., F.A.C.P., Associate Professor of Clinical Medicine, Washington University School of Medicine, St. Louis; and HARRY C. BALLON, M.D., C.M., F.A.C.S., formerly Assistant Professor of Surgery, Washington University School of Medicine, St. Louis. Large 8vo. Pp. 1070, with 637 illustrations. 1935. London: Henry Kimpton. 65s. net.

Vorträge aus der praktischen Chirurgie. Edited by Prof. Dr. ERICH LEXER, Direktor der Chirurgischen Universitäts-Klinik, München. Part I. *Die pyogene Allgemeininfektion und ihre Behandlung*, by Prof. Dr. ERICH LEXER. Roval 8vo. Pp. 31. 1935. Stuttgart: Ferdinand Enke. RM. 1.60.

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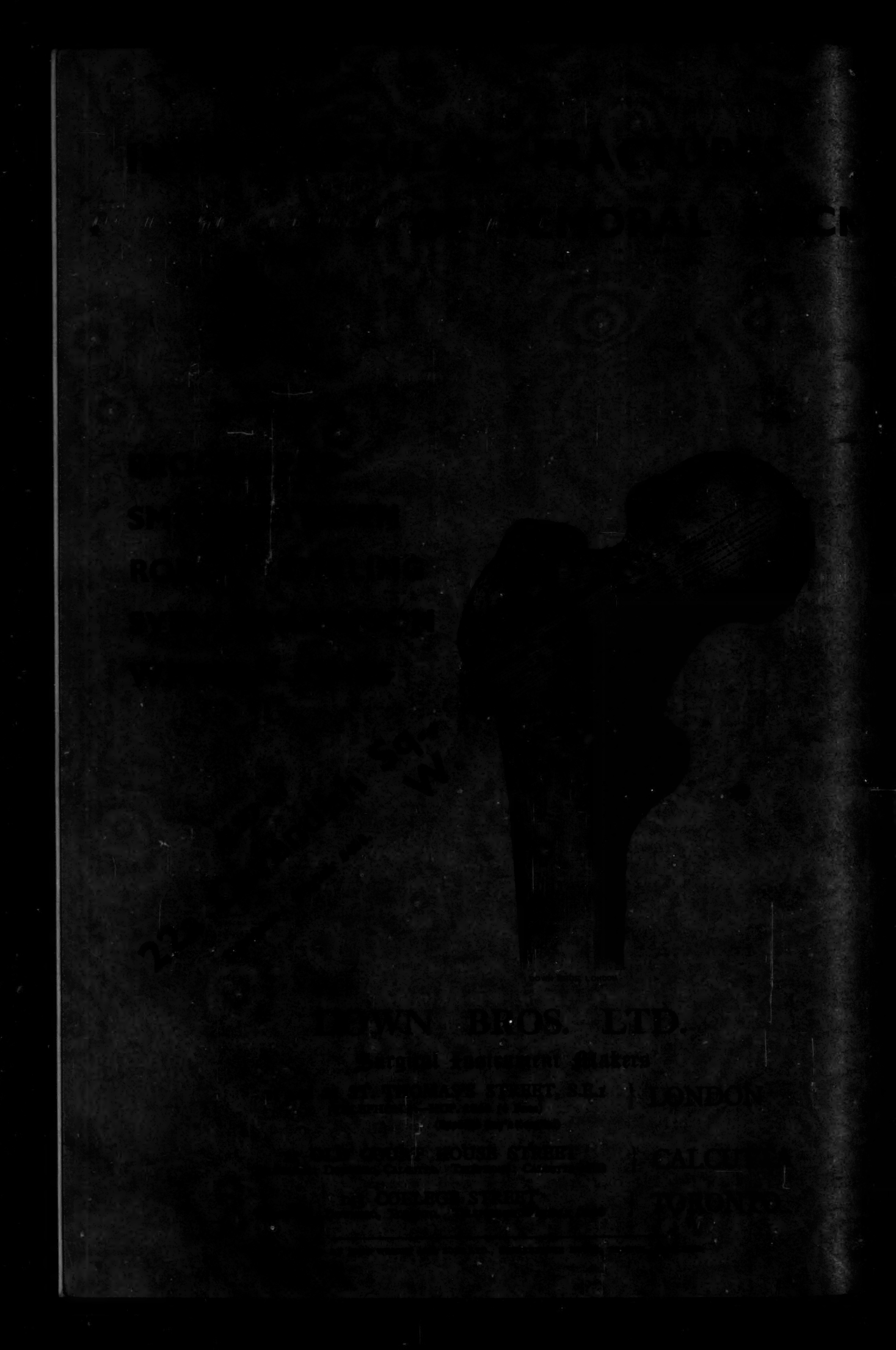
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